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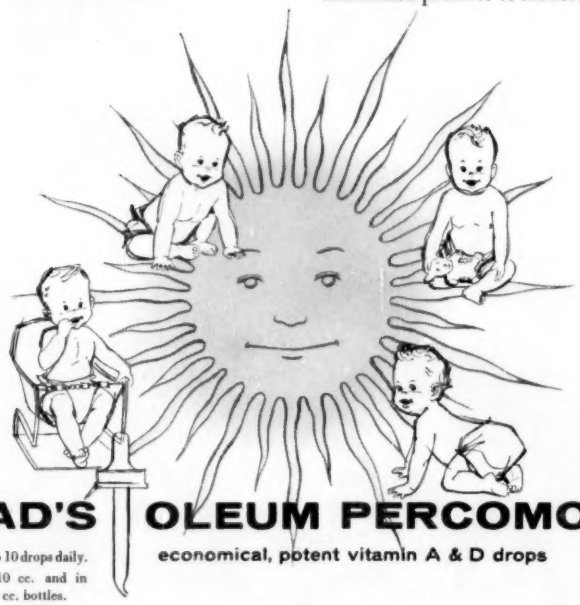

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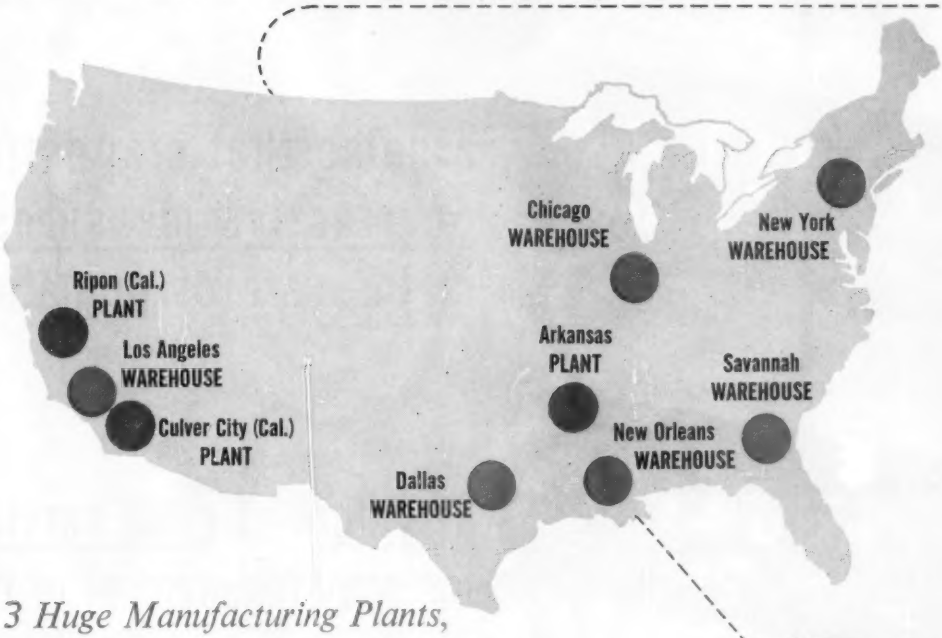

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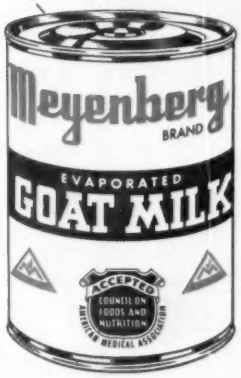

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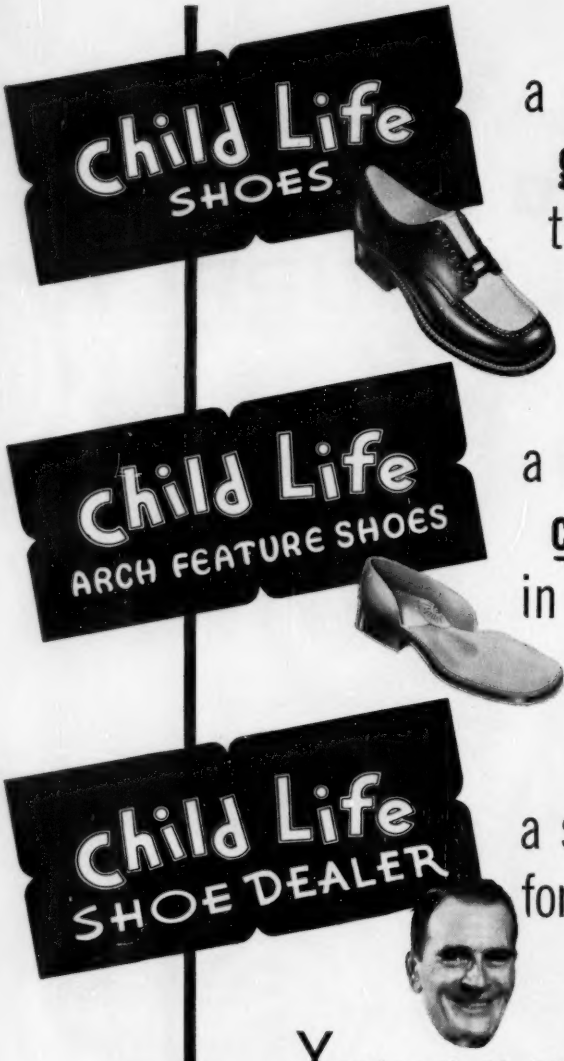

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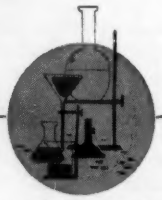

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LETTER FROM THE EDITORS

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It has been felt, in line with the policy of the Editorial Board to attempt to keep abreast of the major interests of all pediatricians by such innovations as the Scientific Exhibits, editorials on timely topics, etc., that some change in the physical form of this JOURNAL was in order. The Editorial Board agrees and feels that the changes made are well conceived and executed. The editors will strive to continue to do their part in selecting new, reliable, and informative material for the readers and in presenting it without undue delay.

CHIEF EDITOR

CLINICAL EVALUATION OF PULMONARY AND THORACIC ELASTICITY IN CHRONIC POLIOMYELITIS PATIENTS

D. L. WATERS, M.D.; DAVID G. DICKINSON, M.D., and JAMES L. WILSON, M.D., Ann Arbor, Mich.

IT HAS been previously shown [1] that the elasticity of the lungs and chest wall is decreased in patients in whom there has been respiratory impairment due to poliomyelitis. This change is of considerable practical importance in the management of such patients, because it increases the work of breathing and decreases the efficiency of mechanical respiratory aids. It further affects the rapidity with which such patients can be weaned from mechanical aids and influences the length of unassisted breathing time, since the latter is dependent upon the work of breathing as well as the respiratory reserve. These changes can explain why similarly involved patients may require vastly different tank pressures for adequate ventilation when frank obstructive factors are absent.

We are reporting an evaluation of a simple clinical technique for estimating the combined elasticity of the lungs and thorax which can be used wherever a tank respirator and a continuously recording spirometer are available. The limitations of the technique and the application of the results are discussed.

CLINICAL MATERIAL

Eight patients were selected for this study, ranging in age from 14 to 36 years. Three were male and five were female. Two had their acute illnesses during the summer of 1952, and the remainder, during the summer of 1953. All but two required at least part-time respiratory aid. Two patients had been in tank respirators during the acute phase of the disease but at the time of this study required no respiratory aid. The vital capacities of the group ranged from about 100 cc. to almost 1,500 cc.

METHODS

The patient was made comfortable in the tank respirator * operating at 20 cpm, and respirations were recorded on the continuously recording spirometer,* using the standard rubber mouthpiece and a nose clip. The tank pressure gradient was then set at —25 cm. of water, and the tidal air volumes were measured. The tank pressure gradient was subsequently reduced to 20, 15, 10, and 5 cm., respectively, with tracings of the tidal air volumes obtained at each pressure. Each pressure gradient was maintained only for a period long enough to obtain an accurate measurement of the average tidal air volumes; the average time required for the complete measurement was about 10 minutes.

In all instances the patient was instructed to relax and to allow the respirator to do all the breathing for him. Six determinations were made on each patient, when possible on consecutive days at approximately the same hour each day. In starting at the higher pressure gradient and successively reducing it, the patients were initially hyperventilated; it was hoped that, by doing this, the lung cohesiveness which develops in patients who have been breathing shallowly for a long period would be overcome. We had also hoped that such a procedure might lessen the tendency of the patient to assist himself at the lower pressure gradients; that this technique was not consistently successful is apparent and accounts to some extent for the wide range of values of tidal air volumes at the lower pressure gradients. The data obtained in this fashion are illustrated graphically by plotting the average tidal air volumes (ordinate) against the negative tank pressures (abscissa). Figure 1 illustrates a typical record and shows the method used to determine the average tidal air volume produced by each pressure gradient. All figures for air volumes represent the actual observations; it was not thought important to reduce volume measurements to standard conditions of temperature and pressure.

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* The Emerson tank respirator and the Sanborn Metabulator were used.

RESULTS

Considerable variation in the tidal air volumes produced by a given pressure gradient was encountered. The Table shows the maximum differences in tidal air volumes found in each patient at each of the several pressure gradients used, and Figure 2 is a graphic representation of the range of values found at each gradient. It is interesting to note that there is no consistent pattern of variation from patient to patient and that artifacts can be found at each of the pressure gradients employed. However, in spite of this variability, the curves do qualitatively

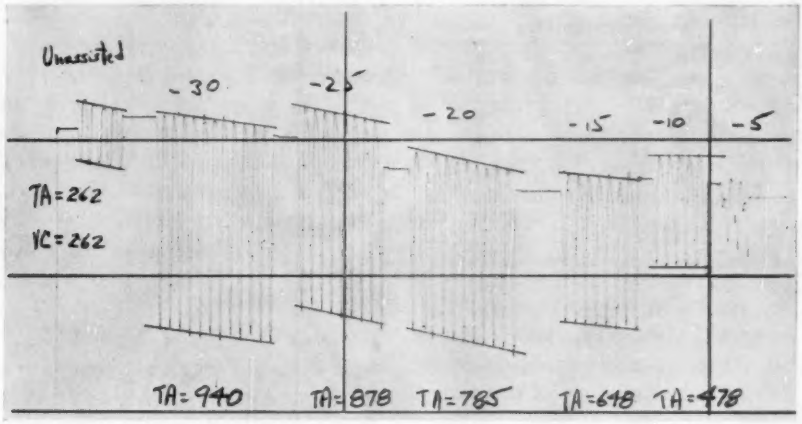


Figure 1.

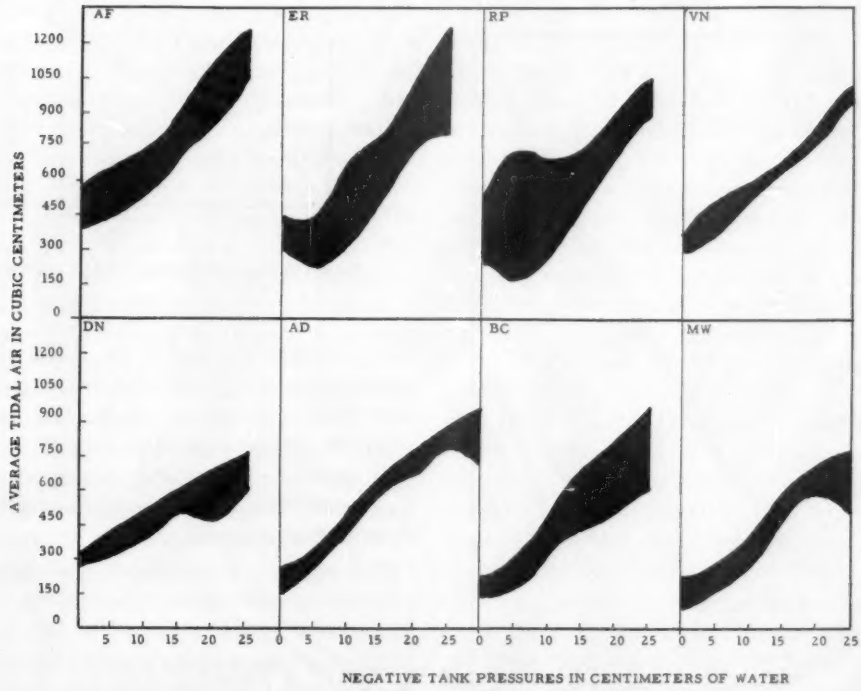


Figure 2.

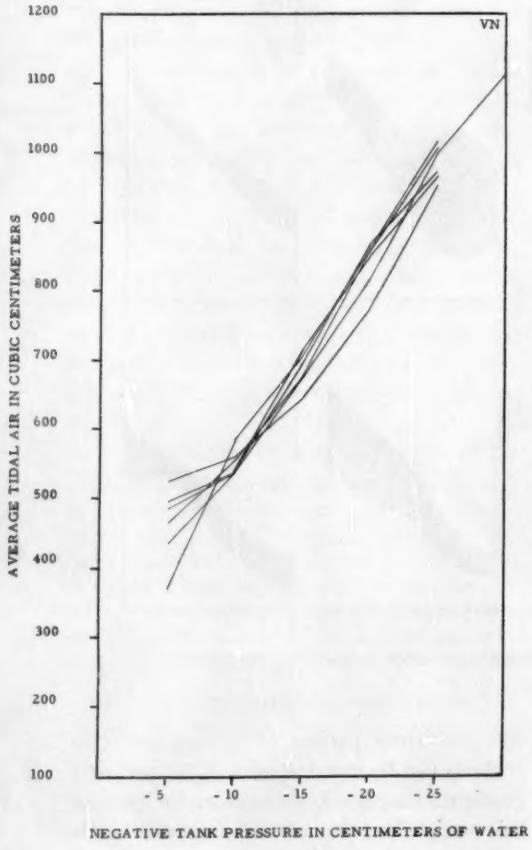
The Differences Between Maximum and Minimum Tidal Air Volumes Obtained at Various Pressure Gradients in Six Separate Determinations on Each Patient*

Patient	Pressure Gradients, Cm. of Water					
	30	25	20	15	10	5
M. W.	...	246	108	92	154	...
B. C.	...	389	361	279	171	115
A. D.	227	90	105	61	78	62
D. N.	...	154	246	96	124	108
V. N.	...	61	93	61	46	155
R. P.	...	150	169	247	401	571
E. R.	...	431	277	262	362	216
A. F.	...	90	248	216	186	263

* These differences are expressed in cubic centimeters.

reflect the combined elasticity of each patient's lungs and chest wall, since it can be seen that there is considerable difference in the over-all slopes of some of the curves. This is particularly evident when the over-all slope of Patient D. N. is compared with

Figure 3.



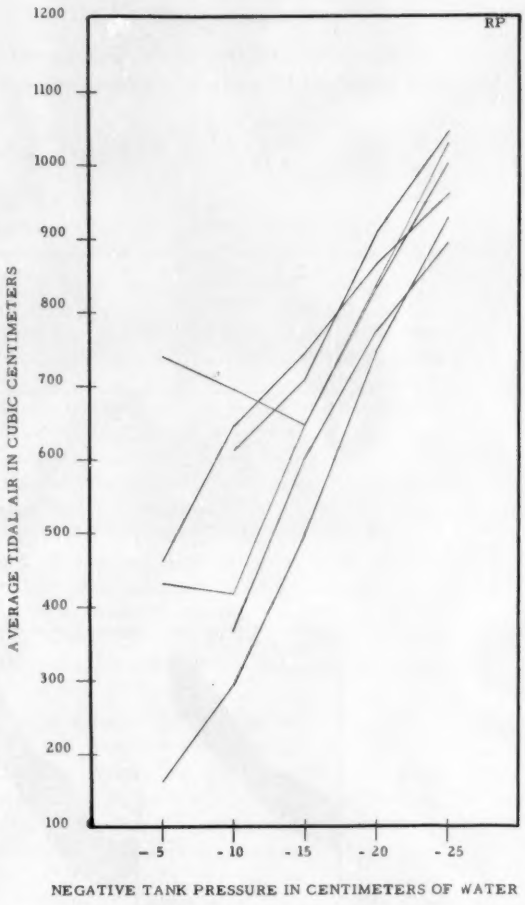


Figure 4.

that of Patient A. F. or A. D. A flat curve results when the elasticity of the lungs and chest wall is decreased. D. N. and A. D. represent two patients with about the same vital capacities whose curves suggest that A. D. has much the more elastic lungs and chest wall of the two.

It is apparent from these results that the pressure-volume curves obtained in this manner are at best a qualitative estimation of the elastic state of the patient's lungs and chest wall. It was virtually impossible to reproduce results from day to day with a consistency which would make this procedure suitable for making any quantitative estimation of the elasticity. One patient demonstrated a remarkable consistency, for

which we have no explanation (Fig. 3). In the group, as a whole, differences between the maximum and minimum tidal air volumes at a given pressure gradient ranged from a low of about 45 cc. to a high of about 500 cc. Figure 4 illustrates the results obtained from one of the more variable subjects; the difficulty of obtaining anything at all resembling consistent results at the lower pressure gradients, i. e., 5 and 10 cm., is well demonstrated. On two occasions it was impossible to get measurements at 5 cm. pressure which could be plotted.

COMMENT

Affeldt [2] and Ferris † and their co-workers have contributed much to our knowledge of pulmonary function in convalescent poliomyelitis patients. Elaborate studies requiring the use of equipment available only in a few research centers have been carried out in attempts to quantitate thoracic and pulmonary elasticity. They have referred to this elastic quality as pulmonary compliance. The method of estimating "compliance" that we have described can be applied anywhere that a tank respirator and a continuously recording spirometer are available. For simplification we admittedly have sacrificed a certain amount of accuracy; however, the results obtained do yield a useful qualitative reflection of the elastic state of the patient's lungs and thorax and will suffice to provide the clinician with answers to important questions arising during the management of the convalescent respirator patient.

Certain errors introduced through simplification of the technique as described by Affeldt [2] and Ferris ‡ are insignificant when compared to the intrinsic errors inherent in making measurements of this kind on a conscious patient. The source of these intrinsic errors lies in variable inherent mechanical and psychological forces peculiar to the patient himself and are operative regardless of the technique of measurement employed.

The mechanical factors are any resistive forces which impede air flow, e. i., the shape, configuration, and patency of the anatomical airways (nasal and oropharyngeal passages, the larynx, and the bronchi). Anything which obstructs air flow, such as a paralyzed vocal cord, a constricted pharynx, or bronchial secretions, decreases the change in lung volume produced by a given pressure. The patient can further create resistance to air flow by voluntary or involuntary closure of the glottis or by pharyngeal constriction.

The psychological factors may also increase resistive forces, and they may be tempered by the patient's ability and his desire to cooperate. His ability to relax and allow the respirator to do his breathing for him influences the consistency of the results obtained. The manner in which the patient approaches the test, whether he considers it just another annoying ward procedure to be tolerated or something which may have considerable bearing on his program of treatment, helps to determine the effect the psychological factors will have. During this study it became apparent that we could fairly well predict the type of curve we might obtain from one of the patients after a few moments of conversation with her prior to the measurements; when her mood was amiable we found her able to relax and the curves were smooth but when in a cantankerous mood she was likely to resist the respirator. If patients have to be coerced to participate, the results are likely to be variable from day to day. Personality is an influencing factor as well, since the patient who "wants to be a good patient" and who is anxious to please is most apt to cooperate to his utmost ability. Further, the patient's ability to suppress his automatic breathing efforts during the test likewise influences the consistency of the results.

Because of the multitudinous sources of error we feel that several determinations should be made on each patient before any statement can be made concerning the elasticity of the lungs and thorax. Once the data are obtained the clinician can predict fairly well what to expect of a given patient when the time comes to get him out of the tank respirator and into a chest shell or rocking

† References 1 and 3.

‡ References 1 and 3.

bed. The patient with a steeply rising pressure-volume curve, indicating an elastic chest wall and lungs, will do well with this type of respiratory aid, while the patient whose curve is flat will do poorly. It should be noted that the vital capacity is not a reliable guide here, since it is dependent upon both the extent of respiratory muscle paralysis and the elasticity of the lungs and chest wall; two patients with the same vital capacity will have different degrees of success in the chest shell or rocking bed, depending upon the degree of fixation of the chest wall or loss of elasticity.

SUMMARY

A simple technique for estimating the combined elasticity of the lungs and thorax in chronic poliomyelitis patients with respiratory impairment is presented, and its application is discussed. The technique described requires only the use of a tank respirator and a continuously recording spirometer.

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CHANGING STATUS OF RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE IN CHILDREN AND YOUTH

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PEDIATRICIANS have been cognizant for some time that there has been a change in the picture of rheumatic fever and rheumatic heart disease in children and youth. This opinion has been based upon their experience in private practice and as staff members of the larger hospitals which usually care for these patients. In spite of this general impression, however, the American literature has published few reports containing facts which substantiate or refute this clinical observation. Some of the facts describing the situation in New York City are presented herewith.

REVIEW OF THE LITERATURE

A review of the American literature from 1940 to the present time reveals 10 reports relevant to this subject. These reports are briefly summarized in chronological sequence.

The Metropolitan Life Insurance Company in 1943 [1] reported that for its industrial policy-holders between the ages of 5 and 24 years in the interval between World War I and World War II, the mortality from rheumatic fever and chronic heart disease declined 70%. In the last five-year period of this report, the mortality rate had decreased by one-third.

Keith and Pequeguat, in 1947,[2] reported data on deaths from heart disease in children and young adults in Canada from 1926 through 1943. They reported that "the most hopeful feature of the mortality figures is the steady decline that has occurred in recent years. A remarkable drop in mortality from heart disease and rheumatic fever for Canada has occurred between 5 and 20 years of age. This is not so marked between 20 and 29 years, and no decrease is apparent between 30 and 39 years." The decrease in mortality is summarized in Table 1, which is, in part, constructed from their data.

In 1948, two reports * were published by the Children's Bureau depicting the changes in the 25-year period 1919-1944. These reports showed that there was a distinct decrease in mortality from rheumatic heart disease and rheumatic fever among white children. This decrease was in the neighborhood of 70% for the age group 5 through 9 years, 64% for the group 10 through 14 years, and 57% for the age group 15 through 19 years. However, the picture was different for nonwhite children. Among the nonwhite group no consistent downward trend was visible, except in the oldest age group, 15 through 19 years; even this decrease was far behind that of the white adolescents in both sexes and amounted to 37% from the period 1919-1921 to the last report in 1944.

TABLE 1.—*Mortality Rates in Children in Canada Due to Rheumatic Fever and Heart Disease**

Age Group, Yr.	Mortality Rate/100,000 Population		% Decrease
	1926	1943	
1- 4.....	11.2	5.6	50
5- 9.....	12.2	9.3	24
10-14.....	19.0	9.8	48
15-19.....	21.1	12.6	40

* Constructed from data presented by Keith and Pequeguat.[2]

In 1950 the National Office of Vital Statistics [5] reported that the mortality rates from rheumatic fever and chronic rheumatic heart disease in 1950 were lower than for 1949. Table 2 represents data in 1950 constructed from their report for the country as a whole, demonstrating the extent of the problem.

From the Bureau for Handicapped Children and the Bureau of Records and Statistics, New York City Department of Health.

* References 3 and 4.

Wolff,[6] in 1951, presented data comparing mortality due to rheumatic fever and heart disease in the United States in 1919-1921 with data for 1944-1945. This report showed that for the white population there was a 70% decrease in mortality in the age groups 5 to 9 years and 10 to 14 years and a 60% decrease in the 15 to 19 age group. For the nonwhite population there was no significant decrease in the age groups 5 to 9 years and 10 to 14 years and only a 25% decrease in the 15 to 19 age group.

Quinn and Quinn,[7] in 1951, reported data from New Haven from 1920 to 1948, inclusive. These data showed that the mortality rates due to rheumatic heart disease in the age group 2 to 24 years appeared to be decreasing both in the poorest living area and in the middle ecological area. No distinct changes in the death rates due to rheumatic heart disease could be seen in the age group 25 years and older in any of the ecological areas.

The Metropolitan Life Insurance Company, in 1952,[8] reported that among its industrial policy holders 5 to 24 years of age the mortality rate due to organic heart disease declined from about 28 to about 4 per 100,000 population in the period from 1911 to 1950. The mortality rate due to rheumatic fever declined from about 7 to about 1 per 100,000 population during this same period.

The ninth report found is that of Wolff,[9] in 1953, for the United States, which com-

TABLE 2.—*Deaths and Mortality Rates from Rheumatic Fever and Rheumatic Heart Disease, United States, 1950**

Age Group, Yr.	Deaths		Mortality Rates †	
	Rheumatic Fever	Rheumatic Heart Disease	Rheumatic Fever	Rheumatic Heart Disease
Under 1	21	.....	0.6	....
1-14	592	260	1.5	0.7
15-24	280	712	1.3	3.2
25-34	411	1,232	1.7	5.2
35-44	532	2,596	2.5	12.4
45-54	150	4,230	0.9	24.2
55-64	60	4,471	0.4	32.8
65-74	70	3,338	0.9	42.3
75-84	60	2,276	1.9	70.4
85 and over	10	812	2.1	168.5
Not stated	1	24	...	....
Totals	2,187	19,951	1.4	13.2

* Constructed from data of National Office of Vital Statistics.[5]
† Per 100,000 population.

TABLE 3.—*Rheumatic Fever and Rheumatic Heart Disease Mortality Rates* of Children Under Twenty Years by Sex and Color, New York City, 1940-1950*

Year	Rheumatic Fever					Rheumatic Heart Disease				
	Total	White		Nonwhite		Total	White		Nonwhite	
		Male	Female	Male	Female		Male	Female	Male	Female
1940	2.7	2.2	2.8	1.6	8.7	13.2	12.6	11.4	20.3	38.9
1941	2.2	1.6	2.6	3.0	4.1	9.6	8.2	9.6	20.8	19.2
1942	1.5	1.7	0.8	2.8	5.2	8.6	7.6	8.3	18.4	18.4
1943	2.3	2.0	1.9	6.7	7.5	9.0	8.6	7.8	16.2	21.3
1944	2.1	2.0	1.9	6.5	2.4	6.7	7.1	5.6	9.6	13.2
1945	2.1	2.2	1.6	3.7	6.9	5.5	5.8	4.5	11.1	6.9
1946	1.8	1.4	1.6	5.9	4.4	4.4	3.5	4.5	5.9	11.1
1947	1.7	1.3	1.4	3.4	6.4	3.6	3.7	2.7	3.4	10.7
1948	1.4	1.3	1.2	2.2	4.1	3.5	3.5	2.8	5.5	8.3
1949	1.6	1.1	1.6	4.2	3.0	3.6	3.2	3.0	7.4	9.0
1950	0.7	0.6	0.7	2.0	0.0	2.4	2.4	1.6	7.2	6.7

* Per 100,000 population.

RHEUMATIC FEVER
MORTALITY RATES OF CHILDREN UNDER TWENTY YEARS
BY SEX AND COLOR

1940 - 1950

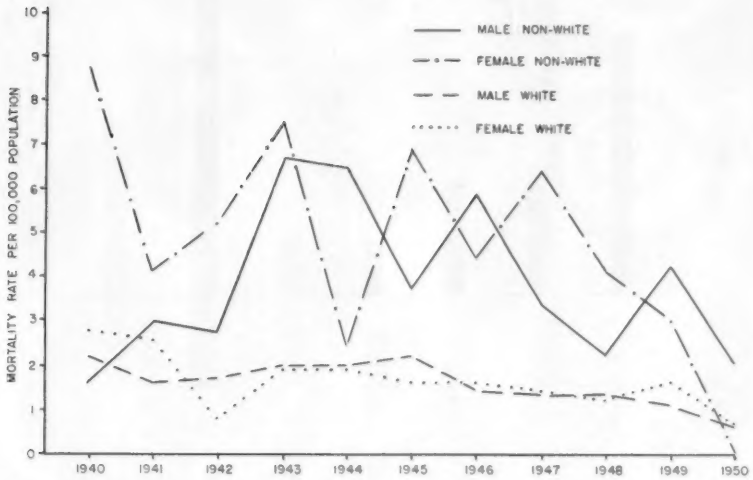

CHART 1

RHEUMATIC HEART DISEASES
MORTALITY RATES OF CHILDREN UNDER TWENTY YEARS
BY SEX AND COLOR

1940 - 1950

CHART 2

pared mortality data in 1919-1921 with data in 1948. This report shows a decrease in mortality among white children of more than 80% for the age group 5 to 9 years, approximately 80% for the group 10 to 14 years, and a little less than 70% for the group 15 to 19 years. However, for the nonwhite group the decreases were approximately 30%, 40%, and 50% in the age groups 5 to 9, 10 to 14, and 15 to 19 years, respectively. Wolff's data show that the Middle Atlantic States have the highest mortality and that New

TABLE 4.—*Leading Causes of Death,* New York City, 1940: Relative Position of Those Causes in 1950*

Cause of death	1940 No.	1940 % of Total Deaths	1940 Rank	1950 No.	1950 % of Total Deaths	1950 Rank
5-9 years						
Accidents	107	26.7	1	66	24.2	1
Heart disease and acute rheumatic fever	52	13.0	2	6	2.2	†
Appendicitis	46	11.5	3	11	4.0	5
Malignant neoplasm ‡	21	5.2	4	66	24.2	1
Pneumonia	20	5.0	5	13	4.7	3
All other causes	155	38.6		111	40.7	
Totals	401	100.0		273	100.0	..
10-14 years						
Heart disease and acute rheumatic fever	124	26.7	1	30	15.3	3
Accidents	67	14.4	2	41	20.9	1
Appendicitis	39	8.4	3	3	1.5	†
Malignant neoplasm ‡	34	7.3	4	41	20.9	1
Tuberculosis	35	7.5	5	8	4.1	†
All other causes	166	35.7		73	37.3	
Totals	465	100.0		196	100.0	
15-19 years						
Tuberculosis	165	23.0	1	36	10.4	4
Heart disease and acute rheumatic fever	130	18.2	2	42	12.1	3
Accidents	72	10.1	3	62	17.8	1
Pneumonia	38	5.3	4	10	2.9	†
Malignant neoplasm ‡	36	5.0	5	45	13.0	2
All other causes	275	38.4		152	43.8	
Totals	716	100.0		347	100.0	

* International Classification, fifth revision.
† Not among five leading causes in 1950.
‡ Includes leukemia and aleukemia.

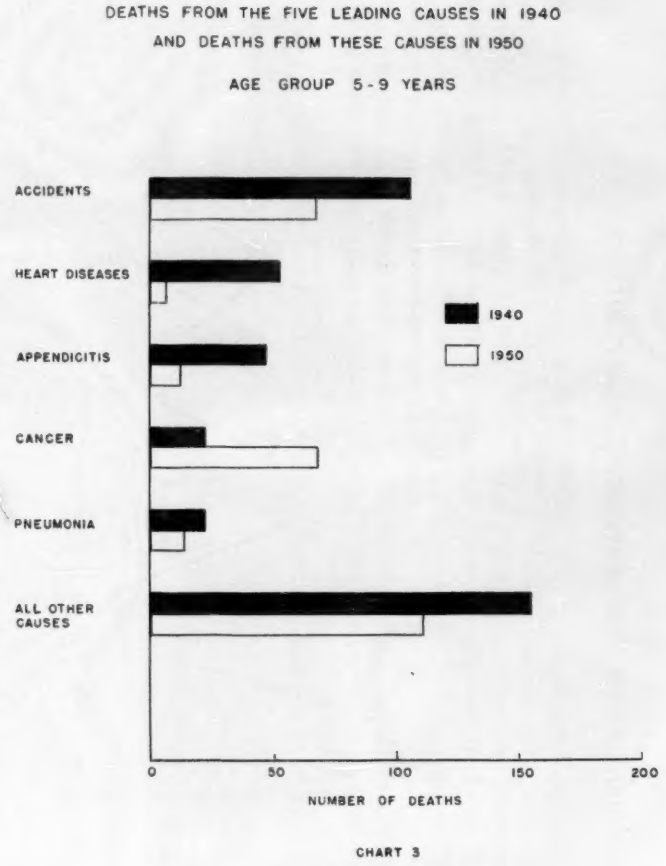

CHART 3

DEATHS FROM THE FIVE LEADING CAUSES IN 1940
AND DEATHS FROM THESE CAUSES IN 1950

AGE GROUP 10-14 YEARS

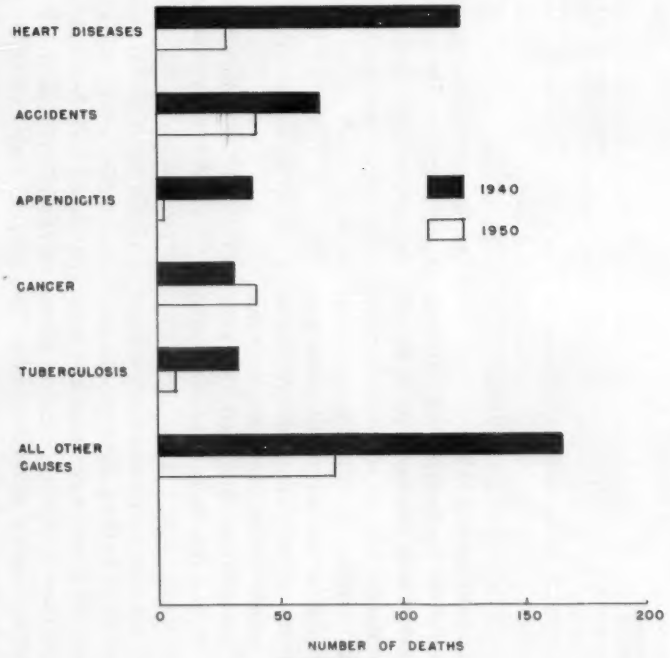

CHART 4

DEATHS FROM THE FIVE LEADING CAUSES IN 1940
AND DEATHS FROM THESE CAUSES IN 1950

AGE GROUP 15-19 YEARS

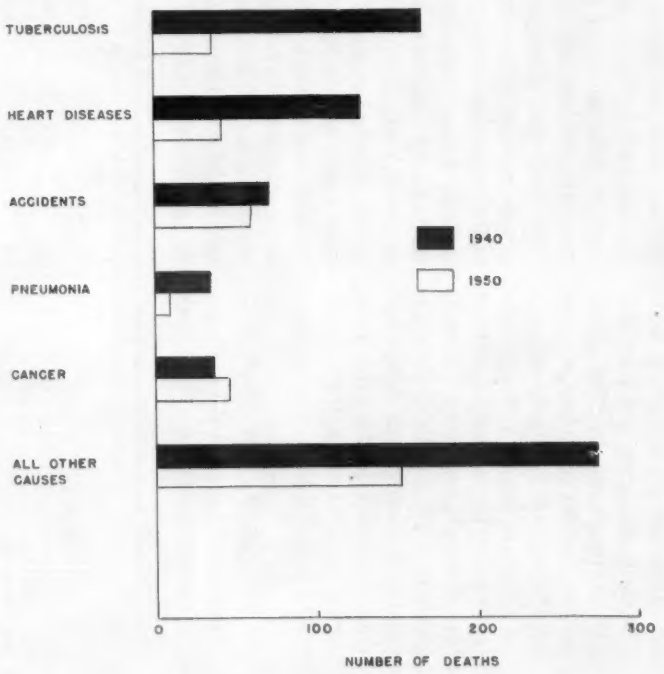

CHART 5

York State ranks seventh highest in death rates due to rheumatic fever and heart diseases, being outranked only by Utah, Delaware, Colorado, District of Columbia, New Jersey, and Pennsylvania, in that order.

The final report is that of White,[10] comparing the relative prevalence of various kinds of heart disease in two series of cases in New England in 1925 and 1950. This report showed that rheumatic heart disease had dropped from first place in the 1925 series to third place in the 1950 series (from 39.5% in 1925 to 23.5% in 1950).

MORTALITY DATA FROM NEW YORK CITY

Table 3 and Chart 1 present the mortality data due to rheumatic fever in New York City from 1940 through 1950. In this 11-year period there has been a 74% decrease in mortality due to rheumatic fever for all children under 20 years of age. The decrease in mortality among the white females was 75%, and that among the white males was 73%. Because of the small number of deaths in each year among the nonwhites, with the resultant instability of the yearly rates, it may only be said that there is evidence of a downward trend.

Table 3 and Chart 2 present similar mortality data due to rheumatic heart disease. In the same 11-year period there has been an 82% decrease in mortality due to rheumatic heart disease for all children under 20 years of age. The greatest decrease in mortality is found in the female white children (86%), with the female nonwhite (83%) and the male white groups (81%) running next in order of decrease. Deaths of male nonwhite children decreased only 65%. In 1950 the mortality rates among the nonwhite groups were still three to four times those of the respective white groups.

Table 4 and Charts 3, 4, and 5 show the leading causes of death in 1940 and in 1950 in New York City in the children's age groups in which rheumatic fever and rheumatic heart disease are likely to be factors. In 1940 heart diseases were second among the leading causes of death in the age group 5 to 9 years; by 1950 they were no longer among the five leading causes of death in this age group. In 1940 heart diseases were first among the leading causes of death in the age group 10 to 14 years; by 1950 they had dropped to third place in this age group. In 1940 heart diseases were the second leading cause of death in the age group 15 to 19 years; by 1950 they had dropped to third place in this age group.

TABLE 5.—*Cardiac Consultation Service Clinics Completed Diagnoses*

Diagnosis	1943		1952	
	No.	%	No.	%
Noncardiac	1,553	37.4	3,358	50.3
Potential heart disease	257	5.2	1,124	16.9
Possible heart disease	776	18.7	859	12.9
Potential and possible heart disease	253	6.1	214	3.2
Total	2,839	68.4	5,555	83.3
Organic heart disease				
Rheumatic	1,057	25.5	644	9.6
Chorea	2	0.0	...	....
Congenital	244	5.9	467	7.0
Scarlet fever	8	0.2	...	....
Other	...	....	4	0.1
Total	1,311	31.6	1,115	16.7
Grand total	4,150	100.0	6,670	100.0

ANALYSIS OF CHILDREN SEEN IN CARDIAC CONSULTATION SERVICE CLINICS

The Bureau for Handicapped Children for a period of years has operated five cardiac consultation service clinics, where practicing physicians and physicians in the Department of Health's School Health and Child Health Services may refer their patients for diagnosis and consultation. Table 5 presents the diagnostic picture of the children seen in

these clinics in 1943 and 10 years later, in 1952.

The major changes in diagnosis in this 10-year interval are as follows: 1. In 1952 there were more children diagnosed with nonorganic types of heart disease and fewer children diagnosed with organic heart disease. 2. In 1952 there were more children diagnosed as noncardiac and with potential heart disease. 3. In 1952 there were fewer children with rheumatic heart disease and with possible heart disease. The percentages of children diagnosed with congenital heart disease in these two series are not statistically significantly different. That there are considerable differences in the children seen in the cardiac consultation service clinics in this 10-year interval is evident. Whether these differences are due to a change in the picture of rheumatic fever or heart disease in New York City or due to a change in the pattern of referral of children to these diagnostic clinics by physicians in the City, or to a combination of both factors, is unknown.

These cardiac consultation service clinics also have the additional function of making the recommendation for the educational placement of the school children seen in the clinics. The differences between the 1943 data and those for 1952 are shown in Table 6. In 1952 a much higher percentage of children was recommended for regular class in the public schools. These differences may likewise be due to the changing picture of rheumatic fever or heart disease in the City or to a change in the type of child referred to the clinics, or both.

TABLE 6.—*Cardiac Consultation Service Clinics School Placement Recommended*

	1943 *		July, 1952-June, 1953	
	No.	%	No.	%
Regular class	999	90.8	5,612	98.4
Special class	91	8.2	54	0.9
Cardiac	..	...	30	0.5
Lowered vitality	..	...	24	0.4
Home instruction	3	0.3	40	0.7
Out of school	9	0.8	..	...
Withdraw	1	0.1	..	...
	1,103	100.0	5,706 †	100.0

* Data from three of the five clinics.
† In addition, there were 269 children who were of preschool age.

COMMENT

It is recognized that any study of chronic disease in any part of the community's population which is based upon an analysis of mortality data alone is not complete and does not present the total picture of the disease in the community. In a sense, it presents only one aspect of the end-result of the disease. To complete the picture, additional data would be necessary which would include the incidence and prevalence of rheumatic fever and rheumatic heart disease among children in the community. These data are usually obtained from a study of admissions to hospitals and convalescent institutions and from a study of the school age and post-school-age populations. These latter studies are usually time-consuming and expensive to perform.

The data presented do indicate a significant reduction in childhood mortality due to rheumatic fever and rheumatic heart disease. The data also indicate some change in the diagnostic pattern of children seen in the cardiac consultation service clinics of the Department of Health, with a higher percentage of nonorganic "heart disease" and noncardiacs and a lower percentage of heart disease which is rheumatic in origin in 1952 as compared with 1943.

Undoubtedly, in recent years, several factors have contributed to this changing picture. The general level of child health has been greatly improved. Improvement in social conditions, in the standard of living, and in nutrition and some elimination of slum areas are all possible factors. Many infections, including those due to strepto-

cocci, are now treated earlier and more effectively by the sulpha drugs and antibiotics. At least some children in New York City known to have had a recent attack of rheumatic fever are receiving prophylaxis, although not the total number by any means. From a public health point of view, prophylaxis is one of the major steps which should be encouraged and promoted in any community which has a relatively high incidence of rheumatic fever. The Bureau for Handicapped Children has already taken initial steps to encourage wider use of drugs of prophylactic value by (1) the allocation of a small amount of funds to purchase a supply of oral penicillin for use by the children's cardiac clinics of three voluntary hospitals in the City and (2) the sending of a copy of the statement on prophylaxis prepared by the American Heart Association to the private physician of each child who has a history of a recent attack of rheumatic fever and who is seen in the cardiac consultation service clinics. The New York Heart Association has currently completed a survey of policies regarding the use of prophylaxis agents in its affiliated hospital cardiac clinics. This survey is the first step in the stimulation of wider use of such agents by these clinics.

There is one other guiding fact which is a result of the data presented in this report. If it is true that there is a decline in the incidence and prevalence of rheumatic fever and rheumatic heart disease in children, certainly this would mean that fewer pediatric beds devoted to this purpose are necessary in hospitals and in convalescent institutions. This fact then should be kept in mind in planning future hospitals and convalescent institutions in the metropolitan area, so that there is not an overprovision of facilities and a wastage of some community funds for this purpose.

SUMMARY

An analysis of data in New York City indicates that there has been a significant reduction in deaths due to rheumatic heart disease and rheumatic fever in children and youth in the past decade. The rate of reduction has been greater in the white group than in the nonwhite. During the same time, the diagnostic pattern of children seen in five cardiac consultation service clinics indicates a similar change, with fewer children with rheumatic heart disease and more children without heart disease being seen. More children seen in these clinics are being recommended for educational placement in regular classes in the public schools, indicating either no cardiac handicap or a cardiac handicap of less severity.

Major emphasis in public health community programs should be placed on early case finding, the securing of an accurate diagnosis of all children having or suspected of having rheumatic fever or heart disease, services of high quality for hospital and convalescent care, good follow-up, appropriate educational placement, consideration of the vocational aspects, and the encouragment of greater usage of prophylactic agents, when indicated, in an effort to prevent recurrences of rheumatic fever.

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METHODS OF THERAPY IN CHILD PSYCHIATRY

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IN PATHOLOGICAL human behavior the action is not the conflict itself but is a token overdetermined compromise expressed in an attempt to attain relief from difficulties causing undue anxiety. To understand the psychic disturbance is to go beyond the deviant behavior itself and to evaluate this through all the channels of communication available, providing information concerning the genetic and dynamic forces affecting the total personality.

In the patient-physician relationship, communication is a basic process in which the patient endeavors to convey his problems to the physician, who, in turn, attempts to understand them and make them more meaningful to the patient. The pathological behavior which is expressed by the patient to the therapist is an effort on the part of the patient to relate his problems and the unworkable way in which he has tried to solve them. This process of communication, which consists of the spoken word, signs, and symptoms, all of them having implicit and explicit qualities, is disturbed by reason of the underlying psychopathology. The patient's facility for communication will be influenced by the severity of his disorder and his innate potentialities for communication. The value of the therapist depends on the specific techniques he is able to use, his skills in understanding what is communicated by the patient, his facility for making the communication meaningful, and his capacity to clarify the problems in accord with the patient's ability to integrate the information in the resolution of them. Here, too, much goes on that is outside of awareness as well as in awareness on the part of the therapist in his relation to the patient. However, with adult therapy the communication is mainly by means of speech, through which are expressed the problems, desires, wishes, and difficulties of the patient and through which the therapist conveys to the patient adaptive information or understanding about the difficulties.

With children, the therapist is beset by other factors in communication. It is obvious that children do not possess the ease to communicate by means of the spoken word, and therefore other skills must be developed to clarify and resolve the behavioral difficulties of the child. Many expressions which to us as adults have a purely conceptual meaning retain for many years in the mind of the child a significance which is not only affective but well-nigh magical. To a child, a word or gesture of an adult or even the child's own thought will evoke all the content of an emotionally highly charged problem, and thus the thought becomes a formidable reality. For a child, commands, desires, resentments, distrust, and other responses may be so undifferentiated that it is difficult to tell when an act or word is used in a context of reality or is confused with other experiences. The development of language as an adaptive function is a slow process, and the form it takes is determined by the stage of development of the child and by the child's basic object relationships—the significant people with whom the child lives. Therefore, forms of language are used in play therapy to permit the child to reveal through the unique language patterns he possesses at the time his basic problems.

Understanding of the importance of play therapy, in which the therapy is anything but "play" in the usual sense of the word, requires the use of an understandable concept of the development of language. Such a

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perspective will singularly facilitate the interpretation of the child's communications. The child's mind may appear opaque to casual adult observation because the thoughts of the child are intermediate between the autistic thinking of infancy and the logical thought processes of the adult. It is a kind of mental activity which the adult has long since left behind or repressed. Briefly, just as the baby is a primary unsocialized person, so too, language is primitive and undifferentiated. The cry or the vocal response expresses the status or need of the baby without regard for object relationships. Quickly, there is developed a nonverbal object relationship in the form of emotional communion to which the term empathy is given. The infant is able to detect whether or not he is in a secure atmosphere pervaded by good feelings. Gradually, with the ability to imitate sounds, he reproduces and imitates the ideas and movements of those around him. However, the thought processes appear to be autistic, that is, in a state of primary unsocialized symbolic activity. In other words, the thought processes are not adapted to reality, are individual and uncommunicable as such by means of the spoken word. Then, as the child is able to construct short sentences, there is the development of egocentric speech, and it is upon this type of language that our primary attention is focused. Up to about the age of 7 years, this egocentric behavior is dominant. In this period, the mind can leap from a premise to a conclusion at a single bound with little value placed on causality and the faculty for explanation. Thinking possesses a magical quality in which images and symbols express the feelings involved, and these feelings may be related to one object as well as to another. Gradually the transition occurs from this period of prelogical thought, which possesses many of the characteristics of dreams, to logical thought and, with it, socialized speech or adaptive communication.

At this period, the plasticity of the child's personality in his responses to his surroundings requires the support of emotionally stable parents to make easier the integration of his own needs with reality. Now, when a problem arises between parents and a child, the behavior is an expression of a disjunctive experience in which at one time there was an attempt at communication with the parents which ended in failure, with the child confused as to his own point of view and that of the other people. Consequently, in the face of deprivations, the child turns the impulses, desires, and needs into symbolic behavior which expresses the desire or wish and the resentment or disappointment. The preoccupation with the difficulty and the anxiety created then restricts the child's personal awareness or contact with reality and blocks the natural growth process. If, on the other hand, satisfactory responses dominate the child-parent relationship and the child is helped to forego of necessity certain immediate impulses with firmness, fairness, understanding, and appreciation of his feelings, this permits personality expansion and draws the child toward constructive communication in object relations.

Since the child communicates his difficulties symbolically, the setting in which therapy occurs should provide a broad range of media for the symbolic expression of the problems. Also, the emotional climate should permit intimacy of understanding between the child and the therapist. Verbal communication alone is futile and already has been tried by parents and well-wishers to no avail, for the child is equipped to respond only in an egocentric context to his problems, and this is beyond the comprehension of the adult. The physical setting therefore contains a wide range of material specifically selected for the use of the child, such as dolls representing various types of humans, toy household furniture and utensils, water, sand, clay, blocks, finger paints, crayons, vehicles, guns, telephones and certain games. Devices which could be used as a resistance to communication are kept at a minimum, and the items are selected to facilitate dynamic action with the expression of constructive and destructive patterns. This material opens up play activity in the areas

which the patient feels especially inhibited or in conflict about.

There is a tendency for most child therapists to keep to a minimum the controlled play activity, by which is meant that the therapist recreates specific problems of the child with dolls. The child also is not permitted free license to indulge in destructive impulses with abandon, and limits are set in keeping with easy and free therapeutic mobility. The method of observation most commonly pursued is to let the child act and talk of his own accord. The child is permitted to interact with the therapist, who responds with interest, a feeling of relatedness, and with understanding, that is, with a therapeutic ear. This opportunity the child has to speak, even symbolically, for himself is a unique experience in human relations for the child. In this setting is observed the manner in which the child's thoughts unfold as the art of asking questions or making statements is directed to get at what is hidden behind the immediate appearance of things, and, as is the case with adults, it is necessary to put aside the defenses before the child lays bare the secrets of the problem and then works on resolving them.

The techniques employed in the playroom will vary and must be refined for the specific needs of the particular childhood disorder. The formulation of the need is based on a detailed developmental history, a detailed account of the present behavior, a history of the parents' present and past life experiences, the observation of the child in a diagnostic play session, psychological testing, and the application of scientific knowledge acquired concerning the dynamic causation of the difficulty. The disorders most frequently seen consist of psychosomatic syndromes, such as nocturnal enuresis, neurotic vomiting, functional headaches, bowel disorders, and bronchial asthma, and character disorders, such as the aggressive-destructive type, the anxiety-phobic type, the schizoid-schizophrenic type, and the regressive-reaction formation type.

Certain basic phenomena will be expressed by a child in the play therapy setting. It is well known that a child may attempt to master anxiety to a specific emotional trauma by the repeated production of the scene through play. Such a tendency becomes more complex in behavior disorders. In play therapy the child may use the original nonadaptive methods of solving the conflict in the attempt to relieve the anxiety. For the most part, however, this material and modifications and distortions of it will be compulsively repeated to the therapist many times as the child seeks help in mastering the anxiety in relation to the difficulties. Nevertheless, the child's communication will vary in many ways, depending on a number of factors. For example, frequency of visits alone is an element which will influence the intensity of the child's responses, with more frequent visits stimulating regressive patterns of behavior. Where the child is seen in a psychoanalytic relationship three to five times a week, the quality of the responses reveal with powerful intensity the impulses and fantasies and the dynamic forces behind them. The age of the child also influences communication. The younger the child the greater the tendency to use nonverbal language. In older children, the interviews tend to approach the talking of adults. Usually, regardless of the age of the child, play dominates the relationship until toward the end of treatment, when talking becomes an integral part of communication. This occurs with the resolution of the symptoms and as the child's adjustment to reality is more secure. The character of the disorder and the skills of the therapist also regulate the responses, as well as the therapist's objectives or goals in treatment. Other factors which should be mentioned are the attitudes and reactions of the parents, very often the father, and the sacrifices the child must make in coming for treatment. The less the sacrifice the more readily does the child enter a cooperative relationship without feeling deprived of his routine pleasurable growth-promoting activities, such as games and companionship of his friends. But where a child does not possess pleasures in living, he avidly seeks the help of the therapist.

A comment is necessary regarding the manner in which parents may influence treatment. To overlook the importance of the parents in any form of child therapy is to invite failure. Without their positive support, treatment becomes untenable. The entire pattern of emotional development and social maturation of the child is determined basically by the parents. The dependency of the child is both biological and psychological and the relationship of the child to the parents or parent surrogates requires a stable and enduring relationship based on affection and mutual need. In treatment, it is important that the parents are able to achieve an environmental equilibrium relatively free from disintegrating anxiety. Environmental factors are included for close scrutiny to uncover difficulties producing the child's problems and to prevent factors which might block healthy development if the environment remains unchanged. Therefore, the active cooperation of one or both parents is necessary to insure a secure therapeutic atmosphere for the child. Most often the parents' experience is basically educational as they live day to day with the child during treatment. Nonetheless, many gain considerable understanding about their own past life experiences which influence the child's behavior and an appreciation of the dynamic process and the principles involved in child-rearing. But, where the parents are not able to improve the environment through appropriate changes for either neurotic reasons or deeply engrained characterological disturbances, psychotherapy or psychoanalysis is encouraged for them.

The therapist must keep himself in proper focus regarding his position and responsibilities in treatment. His every response must be in the service of the problems involved in the parent-child relationship, and his own unconscious needs must not interfere with the procedure. It is a difficult and frightening thing for parents to entrust themselves and their child to the hands of a stranger. It is also extremely uncomfortable for a parent to see the child develop an intimate and eager relationship with the therapist. Also, for the child, such an experience is seductive, causing him to be unsure of his loyalties to the parents or to the therapist. But with the therapist's attention focused on the real business at hand without getting involved in extraneous matters, the parent and child quickly engage themselves in the problems as they arise in treatment. The therapist in this relationship is much more active than in adult psychotherapy, as he shows sympathy for the child's difficulties, shows awareness of the child's negative attitudes, which he helps to verbalize, intercedes with the parents on behalf of the child when it is therapeutically indicated, and often shares with the child new experiences which replace the old. The child recognizes the helpfulness of the knowledge the therapist possesses and turns to the therapist as an ideal which he sets for his own behavior in handling problems well. Treatment, aside from aiding the child to master the traumatic emotional conflicts which created the problems, also becomes an educational experience in the problems of living.

Specific methods of psychotherapy may be separated into three groups: child psychoanalysis, free or spontaneous therapy, and controlled or standardized therapy.

During the 1930's, the controlled or standardized psychotherapy was introduced and used to treat especially symptoms of short duration as a means of desensitizing the child to his specific fear. Levy called his method "release therapy." Conn described the procedure as "the play interview," and Solomon, stimulated by the work of Conn, defined his procedure as "active play therapy." All have in common a technique in which the therapist chooses in advance the material to be used and the specific situation the child is encouraged to react to.

Basically these methods are valuable projective tests which in a spectacular and startling way furnish insight into specific problems of the child. They also have been valuable in the field of research in child behavior. However, these methods have been discarded as an exclusive method of therapy, and only occasionally are they used by the

therapist so that he may gain "a quick look" into a specific problem. There is danger in these methods, for they seduce the child to reveal his feelings prematurely, thereby mobilizing intense anxieties because of the fear that the thought may bring about the act or because punitive action may result. The resistance which results may be very difficult to handle. At present, when the specific symptom is of short duration and for the most part uncomplicated by deep-seated family difficulties, the procedure most commonly followed is the spontaneous or free play modified to meet the situation. The therapist picks out the productive material the child presents which reflects the obvious lines of disorder, and the child tends to confine himself to material in play reflecting his problem. This approach also is used where there is a limited working time to engage in therapy.

As for play analysis, this procedure is used sparingly in this country but more so now than in the past. It is a difficult, costly, and time-consuming procedure, used when the patterns of behavior are rigidly engrained in the child's personality. Child analysis has been influenced primarily by Melanie Klein and Anna Freud. Klein's technique is one in which the child's play is considered the same as uncensored verbal communications of adults and the symbolic representations of unconscious content. She applies a method similar to adult analysis, focusing attention on the transference situation and the resistances and interprets the behavior directly to the child. The role the parent assumes in this procedure is limited. The analytic technique is somewhat modified but not the principle.

Anna Freud's procedure modifies the classic technique of psychoanalysis. She does not treat all play responses as unconscious, feeling much of it has conscious content. She also does not feel that the child develops a complete transference situation as observed with adults, but only a partial one. In much of what occurs in treatment, the relationship for the child is a new one with a different person, rather than the unconscious fixed response as if solely to parents. She also makes more use of information from parents concerning the current happenings in the child's life and stresses the educative aspects of the treatment relationship. Her procedure is based on the knowledge that the child's character is in a developmental continuum and that it is dependent on wise adult guidance for the development of values to live by socially. The treatment is recommended where the personality disturbance is severe, with undue neurotic regression and danger that the neurosis permanently blocks personality development.

Regarding the free or spontaneous therapy, consideration must be given that developed by Allen, whose philosophy and procedure influences treatment in many child-guidance clinics. He presents a relationship to the child in which the child can identify with the therapist and stresses that this relationship contains the essential dynamics of normal growth. The therapy permits "a sense of relatedness to another" and at the same time permits the child to "come to perceive and accept his own self as separate and distinct." It is his belief that difficulties arise out of the interference with the normal growth process, and he directs his therapeutic aim toward reviving this interrupted growth process and toward helping the patient "to be himself." In this approach there is conscious neglect of the dynamic understanding of causation, with, instead, emphasis on the concept that normal growth occurs if the interference is removed. Emphasis is placed on altering parent-child relationships so that the child can still live with the parents but be separate in terms of pursuing his own developmental destiny. The treatment is aimed at improved family relationships and developing the capacity of the child to effect harmonious relationships with the realities of living through the experience with the therapist. The child is provided "a growing relationship with the therapist" instead of remaining "an undifferentiated part" of the mother.

The free or spontaneous therapy which is used most commonly today consists mainly

in the application of psychoanalytic principles to the psychotherapeutic relationship. Modifications of technique are based on the individual skills and other therapeutic concepts of the therapist. The objective in treatment is the transformation of the child's deviant disjunctive behavior into constructive egosyntonic adjustment. This begins in the physician-patient relationship, with the communication process directed toward this end. When the word egosyntonic is used, it means that the two persons, the child and the therapist, become attuned to the problems at hand, either in awareness or covertly, and effectively meet the challenge to understand and master that which is creating conflict in the child. This then permits the child's healthy growth patterns to develop. The communication is a subtle interchange of thoughts or opinions in a shared relationship, out of which comes the child's ability to handle his problems more effectively.

Many factors operating in psychoanalytically oriented psychotherapy already have been considered. Still other factors influence this type of therapy. Usually the child is seen once a week, with the parent seen at the same frequency. This procedure is applied to a large number of problems, with treatment demanding flexibility to meet the needs of the specific child. Where the child is very young—approximately 3 years of age or less—treatment usually is through the mother, who is eager for advice in methods of training and in understanding and handling the symptoms. As mentioned, when such environmental manipulation through the parents is not sufficient because of their own neurotic difficulties, treatment for the parent is recommended.

The goals of treatment in this framework are the resolution of symptoms based on an appreciation of the unconscious factors operating to produce the problems. However, it is not expected as in intensive adult and child psychoanalyses that the derepression will be so intense as to reveal directly to the child the primitive highly charged forces of hate and love operating. Instead, there is a strengthening of the ego structure, through partial insight, through catharsis, and through a new situation in which to work on and overcome the anxiety related to the problems. In other words, there is a strengthening of the child's adjustment to his world of reality, with the development of more efficient defenses.

Interpretations are directed early in treatment to the child's unhealthy defensive patterns and later to the actual forces operating to cause the difficulty, when the evidence is clear and the child is able to accept it without overwhelming anxiety and when the wish to get well is strong enough to give him courage to view the facts about himself which he has believed to be unacceptable. That which the parent communicates in therapy, as it is relevant to the child's problems, is revealed to the child in a constructive way when in the play session it is felt the child is receptive to it. However, the reverse does not hold true. The communications of the child are not directly revealed to the parents, and the child's trust is kept. Indirect methods of communication are used with the parents to influence the environment favorably.

Other procedures which stimulate wholesome growth patterns are planned for the child as he is able to accept them, such as special tutoring, special recreational facilities, camps, and other group activities. These he fully enters into as his adaptive resourcefulness improves. The length of time of treatment varies from several sessions to many months, depending on the severity of the child's difficulties. In certain deep-seated character disorders, as with the schizoid-schizophrenic group, treatment may require one or two or more years. If treatis not effective, one must gauge the factors operating in the home which may be undermining the treatment or the procedures used which are blocking therapy. The objective of therapy is to remove the unsatisfactory patterns of behavior and permit the child to have personally satisfactory and socially integrative experiences which stimulate creativity and healthy personality growth.

DIAGNOSTIC LARYNGOSCOPES FOR PEDIATRICIAN AND FAMILY PHYSICIAN

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THERE are a good many laryngoscopes available for infant and child laryngoscopy. However, those that are available have been designed for and by operators who are trained endoscopists. Repeated inquiries by pediatricians and former residents have made it apparent that an instrument designed for inspection of the larynx and for performing the commoner instrumentations with a maximum of safety and minimum of technical difficulty would fill a real need in the armamentarium of the men who normally see the patient first.

Accordingly, construction of such an instrument was undertaken two years ago. The final result is a set of tubes of proper length and diameter. The tubes have a much shorter tip than those now available; thus the laceration of the upper alveolar ridge is minimized. The tubes are closed so that the tongue does not slip into the visual field, a problem that bothers the occasional laryngoscopist. The handle is in the reverse position of the conventional laryngoscope, as experience in teaching the art of endoscopy has shown that the technique of Israel is learned with ease by the newcomer, while the older Jackson technique takes time and remains, even in the hands of the best operators, more difficult for the patient to tolerate. To minimize the bulk of the equipment, the battery is contained in the handle, thus eliminating the separate battery box, wires, etc.

THE INSTRUMENT

The laryngoscope, or more properly the set of laryngoscopes, consists of three blades or tubes and a handle containing the batteries (Fig. 1).*

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* The set of laryngoscopes is available to the profession at Welch Allyn, Inc., Skaneateles Falls, N. Y.

The blades measure 11 cm., 12 cm. and 14 cm. in length for the newborn, infant, and small child up to four or five years of age, respectively. These blades are round and closed, with a short open lip for lifting the epiglottis and exposing the vocal cords. The lip on the tube for newborns is only 0.8 cm. in length; the lip of the tube for infants is 1.2 cm. in length; the child's size scope has a lip of 1.6 cm. These short lips at the end of the closed tubes give full exposure of laryngeal structures, yet keep the tongue out of the field.

The diameter of the tube or blade is 5 mm. for the newborn and premature, 7 mm. for the infant, and 8 mm. for the child's size scope. These dimensions are smaller than those of other scopes. The sizes have been chosen on the basis of actual experience and give maximum ease of exposure with a minimum of instrumental manipulation. The blade for newborn and premature infants is actually the only laryngoscope blade available with a diameter which can be used without undue trauma in a truly premature infant.

TECHNIQUE

The experienced endoscopist will have no trouble using the laryngoscopes herein described. However, as the instruments described are designed for use by physicians who are not trained endoscopists a short review of the technique may well be helpful.

The instrument is held in one hand, and the patient's head is extended as shown in the schematic drawing (Fig. 2). The thumb of the opposite hand is placed over the alveolar ridge, and, as the instrument is inserted, the thumb is used as a fulcrum. The initial insertion carries the blade along the midline until the uvula is seen. With the thumb used as a fulcrum beyond this point, the tongue is then pried upward until the epiglottis is seen. The blade is then dipped below the epiglottis, and it, in turn, is lifted up out of the field of vision (the stippled area in Fig. 2). Once the epi-

glottis has been lifted, the arytenoids and cords appear at the end of the laryngoscope.

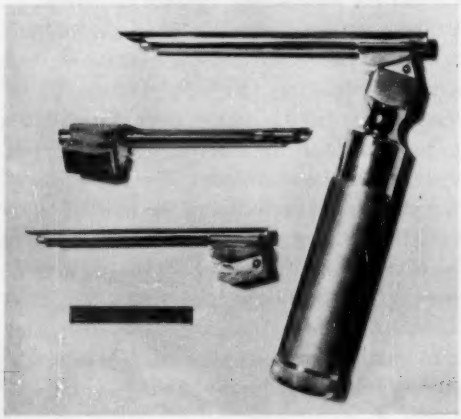
A trick [1] previously described by the author which will be found helpful with the still nursing infant is first to allow the infant to suck on the tip of the gloved finger. A quick, deliberate, and well-aimed substitution of the metal blade of the scope for the finger will often result in good exposure of the larynx before the infant becomes aware of what has happened.

Another point in the technique which is often not completely appreciated by the novice is that the larynx in the premature and newborn infant is relatively higher on the back of the tongue than it is a few months later in life. Thus, if the scope is in the midline and the larynx is not easily exposed, often it is because the end of the blade has completely passed the laryngeal structures. Under these circumstances, especially in the premature and debilitated infant, the esophagus may stand open in front of the advancing scope. If the lumen appears open, is round, and lacks formed structures, the larynx has been passed.

COMMENT

The instruments described have been designed to fill a place up to now left vacant. While they are not meant for the more complicated procedures, they do make it possible for the examining physician to ascertain on the spot the condition of the larynx, and this in a case of croup may make possible a lifesaving decision. The ease of technique makes emergency laryngotracheal aspiration available with suitable suction apparatus,[2] even in the home. And while its use in the removal of foreign bodies from the throat is limited, the laryngoscope could be used for such in the absence of a more complete armamentarium.

Fig. 1.—The three laryngoscopes with universal hook-on battery-containing handle.



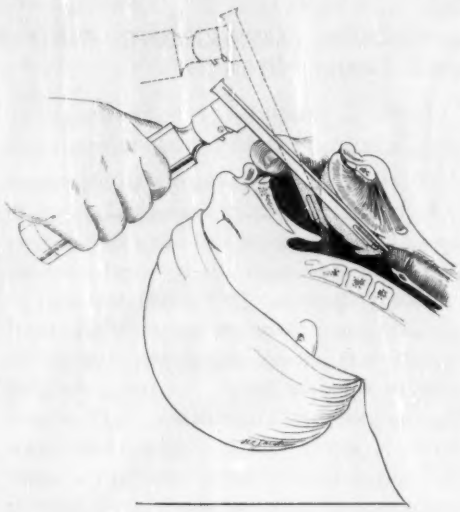


Fig. 2.—Schematic drawing of laryngoscope in use. The instrument with broken lines is in the first position; the instrument with solid lines is in the second position, as described in the text.

SUMMARY AND CONCLUSIONS

A set of laryngoscopes especially designed for use by pediatricians and family physicians is presented.

The instruments have been found satisfactory for the purpose for which they were designed, i. e., the emergency examination of the larynx, laryngotracheal aspiration, and certain other procedures.

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PAROXYSMAL MYOGLOBINURIA

Description of a Case and a Review of the Literature

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PAROXYSMAL myoglobinuria is a disease characterized clinically by muscle pain, fever, and the excretion of dark urine. Pathologically, the disease is a result of an apparently spontaneous muscle injury with the excretion of muscle hemoglobin (myoglobin) in the urine.

MYOGLOBIN

The properties of myoglobin and its occurrence in man have been thoroughly reviewed in a recent monograph by Biörck.[1] This includes the chemistry and physiology of myoglobin. Myoglobin is a hemoglobin in the sense that it is a complex compound of protoporphyrin, ferrous iron, and globin. A comparison of the two pigments follows (data from Biörck [1]):

	Hemoglobin	Myoglobin
Molecular weight.....	68,000	17,500
Fe per molecule.....	4	1
Absorption spectrum		
Visible.....	577	582
UV.....	416	408
pH.....	6.78	6.99
Affinity for oxygen...	Less	More
Renal threshold.....	100 mg./100 cc. plasma	20 mg./100 cc. plasma
Occurrence.....	15 gm./100 cc. of blood	1-3 gm./100 gm. of muscle

The differences in the molecular weight of hemoglobin and myoglobin probably explain the low renal threshold value for myoglobin. This low renal threshold for myoglobin in turn probably explains why we find the greater part of the pigment in the urine rather than in the plasma in those pathologic states in which myoglobin is mobilized.

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For over 200 years investigators have questioned the origin of the red color of muscle, suspecting that diffusion of blood pigment into muscle was an inadequate explanation. Myoglobin is a respiratory pigment occurring in varying amounts in all types of muscle fibers and normally is not free to circulate. It is a protein of muscle sarcoplasm, there being about one-fourth as much myoglobin in the body as hemoglobin. Myoglobin shows a marked affinity for oxygen, and any variations in temperature, pH, and salt concentrations appear to be of less significance in myoglobin than hemoglobin. Myoglobin is by no means the only iron-containing component of muscle; cytochrome, cytochrome oxidase, peroxidase, and catalase containing iron are others.

White muscles are used in repetitive activity and are not called upon for powerful or sustained activity; they have little if any myoglobin and much cytochrome.

Muscles used in powerful sustained contractions, such as the extremities of horses, the heart in large mammals, and the gizzard of hens, require large quantities of myoglobin. This pigment appears to be an oxygen reservoir for use in the recovery or regenerative phase of muscular contraction. It has a great affinity for oxygen and has a rapid oxygen dissociation, permitting quick loading and unloading of oxygen; in this way it serves as a transport agent, although it is a bound and not a circulating pigment.

Important factors which influence the myoglobin level of muscles include exercise and altitude. When a motor nerve to a muscle is sectioned, there is a decrease in myoglobin content.

In 1921, Günther [2] reviewed observations, including his own, on the subject. He is credited with introducing the term myoglo-

bin. Carlstrom [3] in 1931, demonstrated that paralytic hemoglobinuria in horses is due to a release of myoglobin when the animals are forced to exercise after a period of rest and heavy feeding.

Paroxysmal paralytic myoglobinuria in horses or equine myoglobinuria is well known to veterinarians. This syndrome consists of a paresis of the hindlegs associated with the excretion of a red to brown-black urine and affects horses that have been put to work after a period of good nutrition and a period of rest in the stable. The musculature, particularly the iliopsoas and quadriceps femoris, becomes edematous and is found to resemble fish meat; the clinical course is variable; there may be complete recovery or contractures or death due to myocardial damage or uremia.

The pigment is found as a coagulated protein in the tubules, and myoglobin is spectroscopically ascertainable in the urine. The relation of a high glycogen content of the muscle with increased lactic acid formation at exercise may be a factor in causing a change in the internal state of the muscle.

Theorell [4] (1932, 1934) successfully crystallized myoglobin and reported its chemical properties, including a comparison of the absorption spectrum bands of oxymyohemoglobin and oxyhemoglobin. In 1945, Drabkin [5] reported the crystallization of human myoglobin. Independently, in 1947, Theorell and de Duve [6] reported the crystallization of human myoglobin. In 1948, de Duve [7] described a spectrophotometric method for the simultaneous determination of myoglobin and hemoglobin in human muscle.

Bywaters and co-workers* have published data on myoglobin in their investigations of the pathogenesis of the crush syndrome. They established that the pigment found in the urine of patients with muscle necrosis from traumatic injuries is myoglobin.

Rossi-Fanelli [18] determined the nitrogen distribution and amino acid composition of pure crystalline human myoglobin and hemoglobin.

Several theories have been advanced as to the etiology of paroxysmal myoglobinuria. It can be hypothesized that it is due to some hereditary factor which in certain circumstances is activated. The suggestion finds support in the fact that there was a familial component in 8 of 17 cases found in medical literature.†

Myoglobinuria is also known to occur under the following conditions: muscle injury, violent exercise, high tension shock, and Haff disease. These pathologic conditions are similar to the spontaneous or idiopathic form but are secondary to some known exogenous factor. In all types there is a release of myoglobin from the muscle, which produces a myoglobinemia, a resultant myoglobinuria, and in some cases permanent kidney lesions.

The similarity of the spontaneous myoglobinuria to the crush syndrome described by Bywaters and Dible [21] is significant in that myoglobinuria is present in both, but in the crush syndrome the production of myoglobinuria is directly related to extrinsic muscle injury.

There is another syndrome, Haff disease, ‡ brought about by an exogenous factor in which the clinical course and autopsy findings are similar. It was observed in 1924 in the region of the Haff, in Prussia. Fever, muscle pain, and dark red or black urine were prominent. Myoglobin was identified spectroscopically in the urine. The syndrome was also produced experimentally in animals by feeding them fish from the Haff and its tributaries. The nature of the injurious material was never determined, but it has been attributed to refuse from cellulose factories. Eleven similar cases were noticed in 1942, among a population near a small lake in Sweden.[33]

Fischer and co-workers,§ in 1947, described four cases of high-tension electric shock in which the patients survived the shock. Myoglobin was identified in the urine. Muscular tissue is an excellent con-

* References 8-12.

† References 14-28.

‡ References 29-32.

§ References 34 and 35.

ductor, and for this reason the injuries are more extensive than the minute injuries visible at the points of entrance and exit of the current. Two of the patients died with uremia in two and six days, respectively. The affected musculature was pale and showed necrotic changes, including loss of cross striation similar to the finding in both the spontaneous and the exogenous forms of myoglobinuria. The kidneys showed tubules filled by a coagulable mass and a histologic picture indicating blocked epithelium.

The possible relation of spontaneous myoglobinuria to a disorder of carbohydrate metabolism has been mentioned by Meyer-Betz,[14] Hed [25] and Kreutzer.[26] Meyer-Betz found no correlation between the attacks and a diet causing acidosis in the case he reported. In two of Hed's cases, abnormal glucose tolerance tests were observed. In all three of his cases (occurring in brothers), the attacks appeared to be precipitated by exercise in the fasting state.

Some writers (Vannotti,‖ de Langen,[22] Belloni [38]) have reported cases with abnormal porphyrin excretion with muscle involvement. Waldenström [39] has noted the similarity of myohemoglobinuria to some cases of porphyria.

Louw and Nielsen [23] noted that myoglobinuria and progressive muscular dystrophy have many similarities. Their patient with myoglobinuria had eight male relatives with progressive muscular dystrophy, and they hypothecate a close relationship with this latter condition, suggesting it to be a chronic less severe manifestation of the basic defect.

De Langen [22] reported a case observed while he was a physician in a German concentration camp. A 37-year-old minister was forced to genuflect repeatedly for one hour and then was beaten with rifle butts and thrown into a cold wet cell. Forty hours later he had unusually severe muscle pain and could not walk or stand. His urine was red-black, positive for hemoglobin, and contained but a few red blood cells. De Langen sneaked a urine specimen out of camp to his own laboratory and demonstrated myoglobin spectroscopically. There was evidence of renal damage, but the patient recovered.

‖ References 36 and 37.

A SUMMARY OF CASES REPORTED IN THE LITERATURE

I have found 16 cases ¶ and with associates have reported a seventeenth [28] of spontaneous myoglobinuria in medical literature. A brief summary of these 17 cases follows.

CASE 1.—Meyer-Betz [14] (1910) is credited with reporting the first case of myoglobinuria. The patient was a 13-year-old boy who complained of weakness, paralysis, abdominal pain, chocolate-colored urine, and epistaxis. Urinalysis showed the presence of methemoglobin. The patient recovered.

CASE 2.—Paul [15] (1923) was the first to describe autopsy findings; he reports a case of a 42-year-old woman who developed fever, chills, joint and limb pain, and diarrhea. She had red urine which showed methemoglobin on spectroscopic examination. Autopsy findings included pale muscles with Zenker's degeneration and pigment in the kidney tubules.

CASE 3.—Günther [16] (1924) reported a case of myoglobinuria proved by spectroscopy in a man aged 54 years, who developed an influenza-like syndrome, with fever, chills, and weakness. This was followed by a dark-colored urine. Autopsy findings included pale muscles with degenerative changes. Günther [2] published his findings on myoglobin in 1921. He reported that he had obtained a porphyrin from myoglobin.

CASE 4.—Hittmair [17] (1925) described a case in a 41-year-old woman who had several episodes of weakness, paralysis, and dark urine. Cardiac and central nervous system changes were observed. Methemoglobin was found in the urine. The patient recovered. One brother was reported to have had episodes of illness associated with brown-red urine.

CASE 5.—Debré, Gernez, and Sée [18] (1934) described paroxysmal myopathic crises with hemoglobinuria in a 2½-year-old girl. They observed a total of four episodes up to the time of their report. Each time the onset was abrupt, with fever and muscle pains followed by paralysis and passage of "bloody" urine. As the red urine, fever, and pain subsided, a flaccid paralysis at rest and muscle spasm during voluntary movement persisted. The lack of anemia and lack of red cell destruction was stressed.

CASE 6—Huber, Florand, Lièvre, and Néret [19] (1938) described a single episode of paroxysmal myopathic crisis with hemoglobinuria. The patient

¶ References 14-27.

was seen by Debré, who had reported a case in 1934.[18] The clinical diagnosis was apparently made too late for spectroanalysis of the abnormally colored urine. They described a crisis, with pain, fever, hemoglobinuria, and contracted muscles, independent of neurological findings. They stressed that they found no evidence of hemolysis.

Case 7.—Millikan[20] (1939) briefly mentioned a patient he had observed who had myohemoglobinuria. He noted the similarity to the disease in horses. The blood potassium rose to twice normal during exercise.

Case 8.—Bywaters and Dible[21] (1943) reviewed the seven previously described cases and described a case in a 24-year-old man clinically diagnosed as acute nephritis. Limb pain, generalized stiffness, and a poor appetite were the presenting complaints. A smoky-red urine was positive for albumin and benzidine and showed erythrocytes on a centrifuged specimen. He died on the seventh day of illness. Histological examination showed the findings seen in the crush syndrome and absence of changes seen in acute glomerulonephritis. At that time the history of muscular weakness and dark urine was obtained for the first time.

Case 9.—C. D. de Langen[22] (1943) described the findings in a 28-year-old male field laborer. The illness lasted six weeks. It was characterized by abdominal pain, skeletal muscle cramps, temporary paralysis of several muscle groups, dark urine, porphyrinuria, and creatinuria. He emphasized the persistent lack of anemia and the absence of any evidence of increased red cell regeneration as revealed by bone marrow biopsy and peripheral blood studies. A brother with the same symptoms had been diagnosed as having porphyria.

Case 10.—Louw and Nielsen[23] (1944) described a case of a 10-year-old boy who had muscular pain and red-brown urine. Oxymyoglobin was identified by spectroanalysis of the urine. There were eight cases of progressive muscular dystrophy in the maternal relatives, all in males, with females as the transmitters. None of these eight had given a history of dark urine. The authors compare progressive muscular dystrophy with myoglobinuria and postulate that the latter may be an acute attack, with progressive muscular dystrophy representing the chronic form of the condition.

Case 11.—Scherwin[24] (1945) described a case in a 38-year-old man who was hospitalized on two occasions with fatigue, muscle tenderness, dark red-brown urine, and eventual muscle atrophy. The urine was positive for albumin and benzidine and had no erythrocytes. Myoglobinuria paralytica was suspected after the urine returned to normal color, but the necessary tests for myoglobin in the urine could not be done.

Cases 12, 13, 14.—Hed[25] (1947) described myoglobinuria in three brothers. In each the attacks of painful muscles associated with dark red urine occurred after exercise on "an empty stomach." Spectroscopic urinalysis in two of the cases showed myoglobin to be present. The urine of the third case was examined during a remission and was negative for myoglobin. The first two had abnormal glucose tolerance tests. Hed suggested that the pathogenesis is insufficient carbohydrate metabolism.

Case 15.—Kreutzer, Strait, and Kerr[26] (1948) presented a case of spontaneous myohemoglobinuria in a 39-year-old man who could voluntarily produce attacks by stooping or assuming a semicrouching position. Weakness, painful muscles, and passage of dark urine occurred. After an attack he could not produce another for at least two weeks. The authors had the opportunity to study the patient while he voluntarily produced an attack. Myoglobin was identified spectroscopically. Blood and urine studies excluded glycogen disturbance, hemolysis, porphyria, and syphilitic paroxysmal hemoglobinuria. The close link between porphyria and myoglobinuria and progressive muscular dystrophy and myoglobinuria was mentioned.

Case 16.—Wissler[27] (1948) identified myoglobin spectroscopically in the urine of a 6½-year-old boy. He suffered muscle pain sufficient to require that he walk in the equinus position. Dark brown urine was positive for albumin and the benzidine test. There was no evidence of hemolysis, and Wassermann and Donath-Landsteiner tests were negative. Three maternal uncles had suffered from muscle-wasting diseases.

Case 17—Schaar, LaBree, and Gleason[28] (1949) reported a fatal case in a 23-year-old man in which renal insufficiency resulted from hydropic degeneration of the proximal convoluted tubules. The myohemoglobin was identified by spectrometry. Significant histologic changes were found in the striated muscles as well as the kidneys. The patient recalled several previous episodes of dark brown urine and muscle stiffness associated with fever. There was a suggestion of familial occurrence in the history.

REPORT OF A CASE

R. A. (831978), a 9-year-old white twin boy, was referred by his local physician with a note stating that the boy had been sent home from school because of an illness which resembled meningitis or anterior poliomyelitis. He had been well until the present illness.

On May 1, 1951, he refused to get out of bed in the morning because of backache. He had had a slight upper respiratory infection during the previous week. He had done strenuous work in the yard on April 28 and had walked from his home in the country to a ball game in town. His mother

apparently attributed the backache to the exercise and insisted he go to school. He was sent home from school but lay down in the road several times, because of inability to walk due to muscle tenderness and stiffness. He complained of thirst and had one emesis but slept well that night. By May 2 he had developed a fever, dyspnea, and chilly sensations. He was brought by ambulance to the hospital May 2, 1951.

There were two additional events in the history which seemed of possible significance at the time he entered the hospital. There was one case of anterior poliomyelitis in his neighborhood. Two or three weeks before admission the boy had been hit in the suprapubic region with a ball. There had been no dysuria, hematuria, or skin bruise.

Physical examination on admission May 2, revealed a normally developed well-nourished boy who appeared lethargic and feverish. He was oriented. His temperature was 103.6 F., pulse rate 110, respiratory rate 40, blood pressure 120 systolic and 78 diastolic. Neurological examination was reported to show positive neck, leg, and bilateral toe signs. Tendon reflexes could not be elicited. Cranial nerves and sensations were intact. Motor power was not satisfactorily tested, but the examiners stated that the patient could move his legs but that he did not want to.

Dullness to percussion in the left posterior chest was reported and a dry cough observed. There was some tenderness in the epigastric region.

Admission laboratory studies showed a leucocyte count of 23,150 per cubic millimeter, with a normal differential count. The hemoglobin was 16.0 gm. per 100 cc. Urinalysis showed a light brown specimen with 1+ albumin, a trace of acetone, sugar 2+, a specific gravity of 1.011, and a pH 5. Sedimentation rate was 76 mm. in one hour. The cerebrospinal fluid revealed no cells, protein 21 mg. per 100 cc., sugar 33 mg. per 100 cc., and sterile culture. Throat culture showed occasional beta streptococci and coagulase-positive staphylococci. Blood culture taken on admission was sterile on the seventh day. Mantoux tuberculin test with old tuberculin was negative. An x-ray of his chest showed a pneumonic consolidation in the midportion of the left lung.

During the first night in the hospital the nurses noted a "dark reddish-black" urine. In view of the mahogany-colored urine and the painful, stiff extremities, a diagnosis of myohemoglobinuria was entertained. Additional studies were done which supported this impression. The urine gave 4+ guaiac reaction and was free from erythrocytes. Examination of the blood revealed there was no evidence of increased regeneration of erythrocytes as seen in hemolytic anemias. Erythrocyte fragility was within normal limits. Serologic reactions were negative. The pigment myoglobin was identified by spectroscopic examination.# Special studies on a specimen of right gastrocnemius muscle obtained by biopsy showed some evidence of degeneration in some muscle fibers.

Urine creatine excretion was elevated, being 1.1 gm. in 24 hours. Urine creatinine was within normal limits, 0.5 gm. in 24 hours. Urine coproporphyrin, 101 γ per 24 hours, was normal.

Progress in the hospital showed gradual recovery.

A pyridine hemochromogen was present in the urine from the first specimen tested on May 4 to May 7; a trace was noted on May 8 and was absent on May 9.

On May 7, during a glucose tolerance test (reported to be normal), it was noted by one observer that the urine changed from dark brown to a normal color.

The patient's muscle stiffness was most prominent on May 4 and became progressively less marked each succeeding day, with some residual stiffness in back muscles as late as May 19.

The albuminuria persisted until May 8, 1951. The urine remained dark until May 8, when it was 1+ guaiac.

Blood chemistries on May 5, 1951, showed a normal blood urea nitrogen of 18 mg. per 100 cc., a normal creatinine of 1.3 mg. per 100 cc., and a slightly increased potassium of 6.6 mEq. per liter. An electrocardiogram was reported as showing no evidence of hyperkalemia.

Additional chest x-rays showed a partial resolution of the pneumonic process by May 8 and resolution by May 18.

Evaluation of kidney function showed a phenolsulfonphthalein excretion test and an Addis count and routine urinalysis to be normal before discharge. He left the hospital May 22, 1951, in complete remission.

A letter from his mother, Oct. 9, 1951, stated that he had had only one episode of stiffness since discharge. No further episode has been reported to us. Attempts were made to admit the normal twin for study, but his mother was not convinced of the need for study of a well boy.

COMMENT ON REPORTED CASES

A review of the 17 cases taken from German, French, Scandinavian, and English publications and the present case shows that in all there was an acute onset, dark red to mahogany-brown urine, pain, and a mod-

Spectroscopic studies were performed by Dr. Samuel Schwartz and associates, of the research laboratory of the Department of Medicine, University of Minnesota Hospitals.

erate to marked degree of muscle involvement which was incapacitating. The pain in the musculature was associated with a loss of strength and progressed even to paralysis in 12 cases. The involvement was not symmetrical. The legs were usually involved; the arms and back muscles frequently were affected; but the muscles of the neck, chest, and diaphragm usually escaped. In six, the equinus position was a prominent feature. In one case even the tongue was affected, and in another the jaws and intercostal muscles were involved. In several, there was evidence of cardiac muscular involvement. One case (Wissler's [27]) resembled muscular dystrophy; the patient climbed up his own thighs in the fashion of a patient with muscular dystrophy. The muscular involvement in myoglobinuria cases often progressed to paralysis and contractures, but unlike progressive muscular dystrophy, remissions occurred in those cases which survived.

One of the cases is very incompletely described, being mentioned in passing by Millikan in his article on myoglobin.

Myoglobin was identified spectroscopically in eight of these cases; however, a ninth case, one of three brothers reported, was tested during an asymptomatic period; in three of the cases first reported, oxyhemoglobin and methemoglobin were found on spectroscopy, but the differentiation of myoglobin was not discovered.

The ages ranged from 2¾ years to 54 years. Six patients were under 16 years of age. Three were females. Ten gave a history of more than one attack.

Autopsies are reported in the four fatal cases; all showed Zenker's degeneration and kidney tubular degeneration.

There was a familial component in eight. Fifteen had albuminuria, and in 11, casts were reported.

Serologic studies and the Donath-Landsteiner test were done in the majority of cases and were negative. There was no evidence of red cell destruction to account for the pigment in the urine.

In most of the cases there was a history of a poor tolerance to exercise.

It is interesting to note the initial clinical diagnoses in these cases:

Acute nephritis	3
Chronic nephritis	1
Poliomyelitis	2
Progressive muscular dystrophy	3
Phlebitis	1
Hemoglobinuria	2
Influenza	1
Nervous breakdown	1
Epidemic myalgia	1
Acute porphyria	2
Upper respiratory infection (rule out atypical pneumonia)	1

COMMENT

Spontaneous myoglobinuria may not be so rare a condition as would appear from the few cases reported. Our own case illustrates how transitory is the pigment in the urine and how easily it could be overlooked.

Reference to the initial diagnoses in the reported cases illustrates the wide variety of clinical conditions which this syndrome may resemble. The spectroscopic proof is not readily available to most laboratories. Owing to the low renal threshold for myoglobin and the rapid rate at which this substance is removed from the blood stream, it is difficult to demonstrate it in the blood. The etiology of paroxysmal myoglobinuria remains unknown.

The similarity to syndromes produced by exogenous factors, including experimental crushing injuries, only clarifies the fact that muscle pigment or myoglobin which is normally not free to circulate is for some reason released from the muscle.

The renal injury, which is a frequent precipitating cause of death in these cases, appears to be the result of tubular injury. This was supported in Case 17 (Schaar, LaBree, and Gleason [28]) by the demonstration of hydropic degeneration of the tubular cells, rather than the finding of an excessive number of precipitated casts in the tubules, as suggested by other writers (Bywaters*). The precipitation of pigment casts in the

* References 8-12.

tubules shown by Bing [40] is more likely the result of a lower renal activity than the cause of it.

SUMMARY

A case of paroxysmal myoglobinuria in a 9-year-old twin boy is presented.

Seventeen such cases previously described in the literature are reviewed.

In the present case the presence of myoglobin was demonstrated by the finding of typical spectral distribution curves.

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INSULIN REQUIREMENTS OF CHILDREN WITH DIABETES MELLITUS MAINTAINED IN GOOD CONTROL

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A STUDY of juvenile diabetics who were maintained in good control was undertaken to determine the exogenous insulin requirements in relation to age and growth and also to ascertain whether there is any relationship between the insulin requirement and the age of onset of the disease. The study is based on serial observations of the insulin and nutritional requirements and of growth of 48 children with diabetes mellitus under continuous management in our clinic. These patients have maintained essentially physiological control of their disease within weeks after onset, and at the time of the observations of this study they were also free from known infections and emotional disturbances.

Our criteria for classification of patients in varying degrees of control have been reported repeatedly in the literature.* They are as follows:

(1) Very good to excellent—urine specimens free from sugar except for very occasional traces; very occasional mild insulin reactions.
(2) Good—urine specimens free from sugar except for occasional minimal glycosuria; occasional mild insulin reactions.
(3) Fair to good—more than one-half of the specimens free from sugar but minimal glycosuria in the remaining specimens; occasional insulin reactions of varying degree.
(4) Fair—less than one-half of the urine specimens free from sugar and varying amounts of sugar in the remaining specimens; occasional insulin reactions of varying degree.
(5) Poor—urine specimens containing varying amounts of sugar continuously; occasional insulin reactions.

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* References 1 and 2.

MATERIAL AND METHOD

The majority of the patients included in this series satisfy Criteria 1 and 2. For the subjects who came rarely under Category 3, only the insulin requirements of that period of management which could be categorized as 1 and 2 were incorporated in the study. Thus, maximum effort has been made to eliminate the influence of transitory factors which alter temporarily the insulin requirement and control of a diabetic child.

Good control was maintained by administering accurate doses of specific types of insulin in relationship to the intake of a complete diet adjusted to compensate for variations in physical activity. The caloric intake of these children was supplied to meet their needs as expressed by their feeling of satisfaction and devised to include a liberal supply of all known essential nutrients.

With few exceptions, hospitalization has been necessary only at the time of first observation in order to reestablish nutritional status and to determine the insulin requirement of the patient. Each child and his family were given detailed instructions for home care. The parents were taught how to keep a daily home record of the progress of the child, which included entries for diet, insulin requirement, amount and degree of physical activity, the trend of emotional patterns, type of intercurrent infections, description of insulin reactions, and the result of urine examinations. This home record was presented for analysis at each subsequent outclinic visit. In general, as soon as the parents and child were secure in carrying out the instructions, the patient returned for evaluation every four to six months. At this time he had a physical checkup, his degree of control was evaluated, laboratory studies were performed, and his diet and insulin were adjusted when indicated.

The patients are divided for sex and into three groups of A, B, and C, in accordance with the year of onset of disease—from 0 to 5 years, 6 to 10 years, 11 to 15 years, respectively (Table 1).

Height and weight measurements were made at the time of first regulation and each subsequent

TABLE 1.—*Boys and Girls of Groups A, B, C, Maintained in Good Control*

	Age of Onset, Yr.			
	Group A 0-5	Group B 6-10	Group C 11-15	Total No. Cases
Boys	6	9	7	22
Girls	7	7	12	26
Total				48

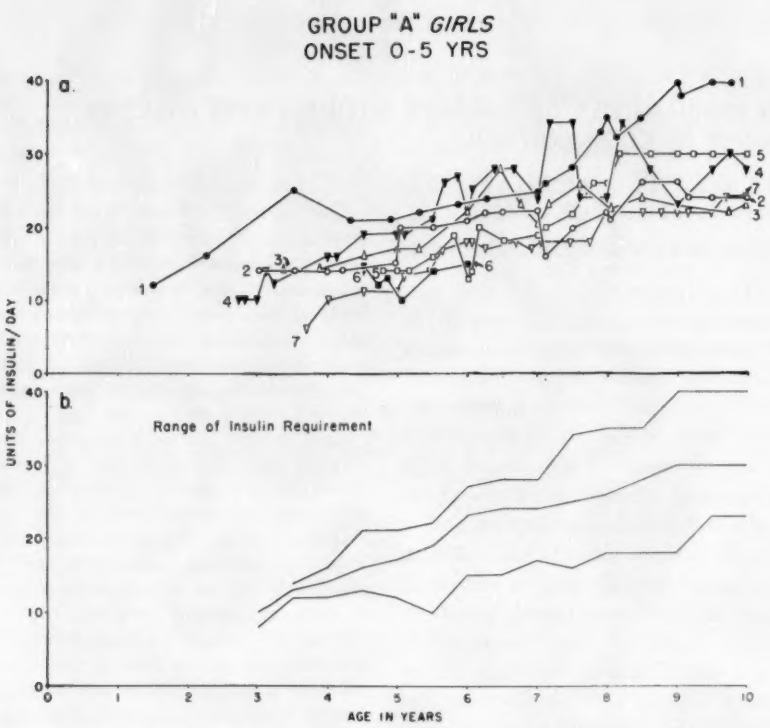


Chart 1.—(*a*) Curves depict the individual insulin requirements of Group A girls from the establishment of good control to the age of 10 years. Numbers designate the individual patients in this group. (*b*) Curves indicate the range of insulin requirements as calculated from the maximal, mean, and minimal requirements from the time of onset to the age of 10 years.

visit. The growth in height and weight of each subject was graphed on growth charts which showed the person's progress in height and weight in relation to height and weight curves of normal boys and girls.[3] The growth of each subject was evaluated similarly to the method used in a previous study made at this pediatric clinic.[1] All the subjects made normal progress in height and weight, which is a further index of the adequacy of the control and management of these patients. The mean values are slightly higher than those of the Iowa growth charts.

The diets prescribed for these subjects were adequate in every respect to maintain optimum growth.

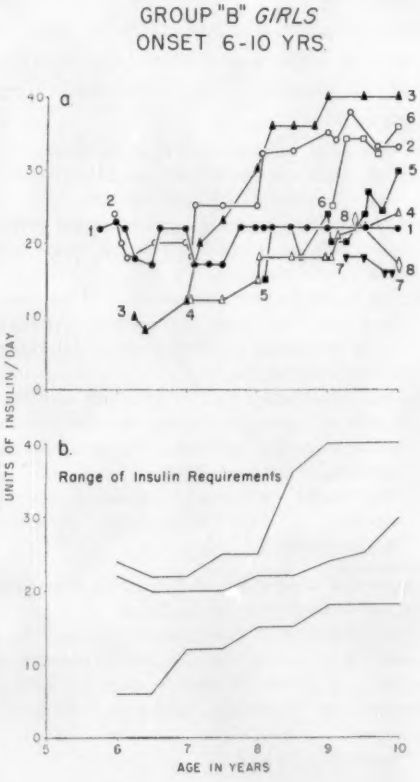


Chart 2.—(*a*) Curves depict the individual insulin requirements of Group B girls from their age of establishment of good control to the age of 10 years. Numbers designate the individual patients in this group. (*b*) Curves indicate the range of insulin requirements of the above Group B girls with maximal, mean, and minimal curves from the time of establishment of good control to the age of 10 years.

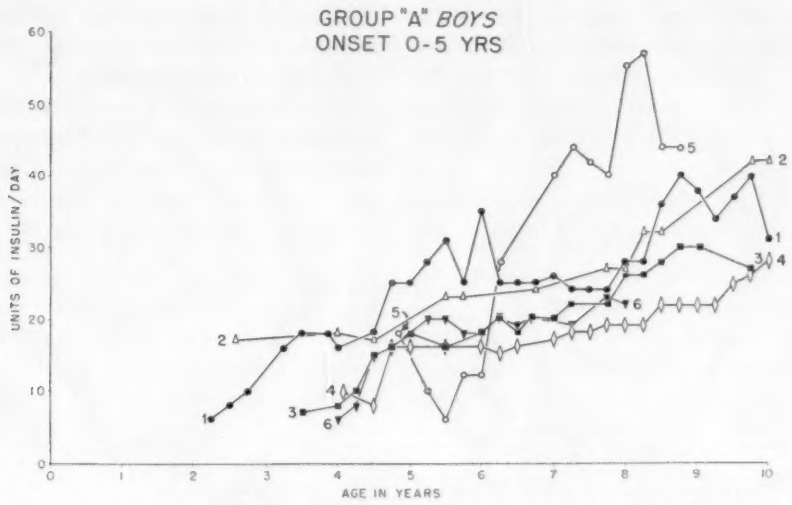


Chart 3.—The individual insulin requirements of Group A boys graphed from their age of establishment of good control to the age of 10 years. Numbers designate the individual patients in this group. Boy 5's insulin requirements are beyond the general range of insulin requirements for his age.

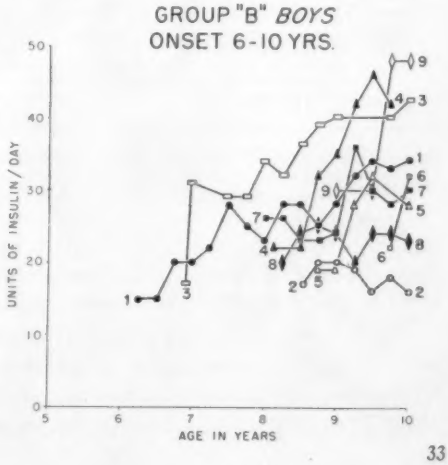
The quantity of protein ranged from 16 to 18% of the total calories as compared to the National Research Council allowances of 10 to 13% of the total calories. The percentage of animal protein furnished by these diets is equivalent to the total protein recommended by the National Research Council. Since a fairly large proportion of the protein in the diets was supplied by milk, the calcium, vitamin A, thiamine, and riboflavin were all supplied in ample amounts. The allowances for the other nutrients, iron, ascorbic acid, and vitamin D, were adequately furnished by other foods in the diet. The caloric content of the diets was individualized for the patient so that growth needs were met, appetite was satisfied, and adjustments were made depending on the amount of physical activity.

In analyzing the data, we have considered first the observations up to 10 years of age and later the observations after 10 years of age. This was done to segregate the characteristics of growth associated with prepuberty.

OBSERVATIONS BELOW THE TEN-YEAR AGE LEVEL—AGE OF ONSET AND EXOGENOUS INSULIN REQUIREMENT

In the curves of daily insulin requirements of girls in relation to their age, as shown in Charts 1 and 2, which are of patients with onset of disease at 0 to 5 and 6 to 10 years, respectively, there is no marked difference observed in requirement due to age of onset. The curves of requirement of Group A either fuse with the curves of Group B or are within the limits of expected standard deviation. Thus, whether the disease began at 0 to 5 years or 6 to 10 years of age, the range of insulin requirement at a given age did not differ materially. A similar finding for boys is revealed in the study of insulin requirement in relation to age as depicted in Charts 3 and 4.

Chart 4.—The individual insulin requirements of Group B boys graphed from the time of establishment of good control to the age of 10 years. Numbers designate the individual patients in this group.



Age, Growth, and Exogenous Insulin Requirement.—To substantiate the hypothesis that the insulin requirement of these children under good control is governed by their growth pattern, the following graphs are presented and evaluated.

Chart 1(*a*), which presents the curves of insulin requirement per day of Group A girls from the time of onset to 10 years of age, gives a vivid representation of their varying needs. For the same group, Chart 1(*b*) depicts the minimal, mean, and maximal insulin requirement. The insulin requirements, which range from 6 to 8 to 10 units in the beginning, are at 10 years minimal 24 units, mean 30 units, and maximal 40 units.

Chart 2(*a*) represents the units of requirement of individual girls of Group B, traced from time of onset to age of 10 years. For the same group, Chart 2(*b*) shows the minimal, mean, and maximal insulin requirements. The requirement at the age of 10 years ranges between 18 and 40 units, with a mean of 30 units. This almost coincides with the curves of Group A.

The insulin needs of boys maintained in good control were studied in Groups A, B, and C, as defined previously.

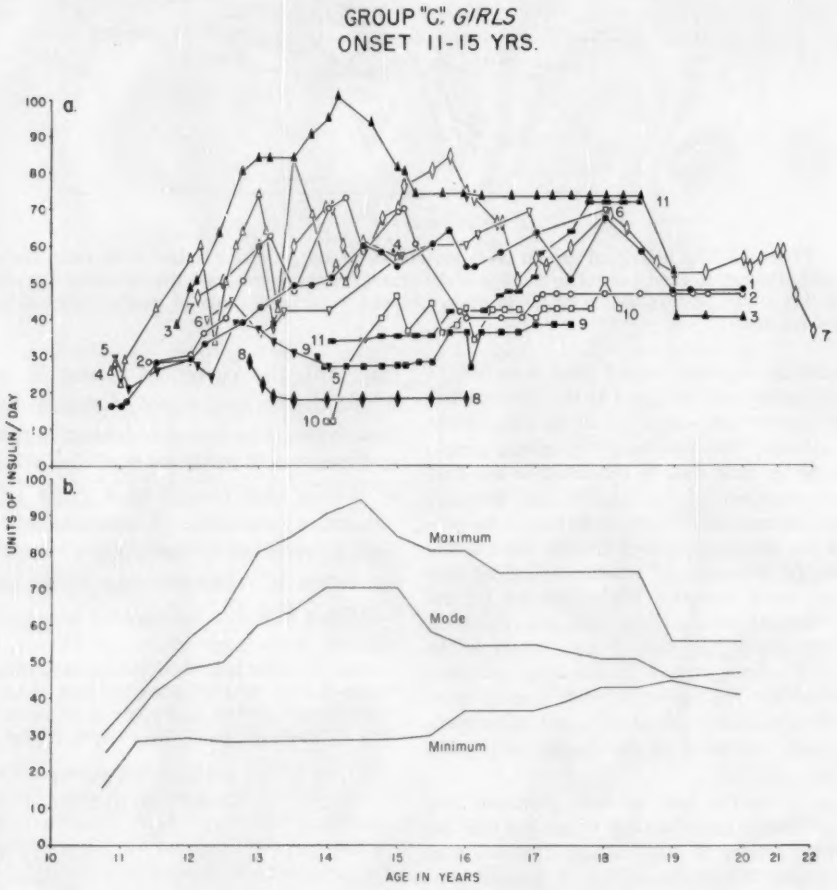


Chart 5.—(*a*) The individual insulin requirements of Group C girls graphed from their age of establishment of good control to adulthood. (*b*) The average insulin curves of Group C girls represent the maximal, intermediate, and minimal insulin requirements from the above Group C girls.

Chart 3 presents the insulin requirement of boys of Group A, individually traced from the time of onset of disease to the age of 10 years. Boy 5 is far above the general insulin requirement. He was under good control and above high normal height and weight since the beginning of therapy. Hence, he was taking above average insulin. The average requirements of the boys of Group A are about 6 units initially and range from 22 to 40 units at the age of 10 years.

Chart 4 is the study of the insulin requirements of Group B boys from time of onset to the age of 10 years, with their individual needs graphically represented. All the boys fall within the range of insulin needs described for Group A boys.

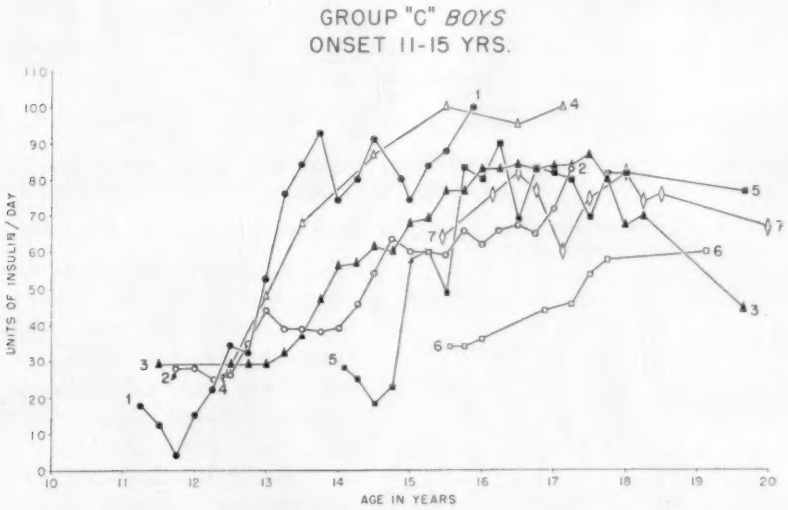


Chart 6.—Curves of individual insulin requirements of Group C boys show their quickly rising requirements assuming their pattern of requirement characteristic of the pubescent age period.

OBSERVATIONS AFTER THE TEN-YEAR AGE LEVEL—GROWTH, MATURATION, AND EXOGENOUS INSULIN REQUIREMENT

Girls and boys with onset of the disease between 11 and 15 years, Group C, are studied in separate groups, as stated before, because of the numerous factors complicating growth at this stage and governing the patterns of insulin requirement.

The daily insulin requirements of Group C girls are represented in Chart 5(*a*). The curves of 11 girls presented here from the time of onset to ages varying from 16 to 20 years give a general picture of requirements with all their variations through this period of growth.

Chart 5(*b*) indicates for Group C girls the maximal, mean, and minimal insulin requirement, roughly represented to show the wide range of fluctuations. Grossly, one can see the requirement in girls rising from 10 to 16 years and gradually reducing to the adult minimums from the ages of 16 to 18 years. This describes only the gross picture of the rise and fall of the requirement. Other aspects will be discussed later, when the charts of group-average insulin requirement are presented.

Chart 6 represents the insulin requirement of Group C boys with the onset of disease between 11 and 15 years. The curves of seven boys are individually traced from the time of onset to the latest date of observation of this study. In the pubescent period, maximal requirements are predicted for both girls and boys.

OBSERVATIONS, TWO TO TWENTY YEARS—ONSET GROUPS COMBINED

Chart 7 represents the insulin requirement of girls under good control at all age periods

of Groups A, B, and C, combined. The insulin requirements of all children are shown from the onset to the final date of observation and are pooled to show the widest possible range and the most definite impression of even minor deviations. The gradual spread C. The insulin requirements show a gradual increase in amount and in spread with advancing age and conform to the pattern of growth of boys.

Charts 9, 10 and 11 trace simultaneously the insulin requirement, height, and weight

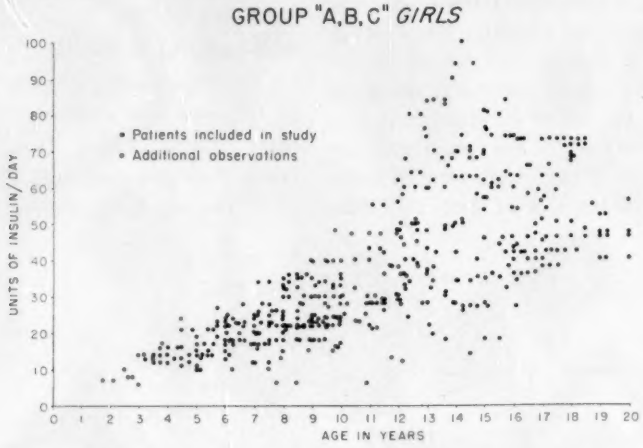


Chart 7.—The scattergram shows the varying insulin requirements of all groups of girls (A B, C) from infancy to adulthood.

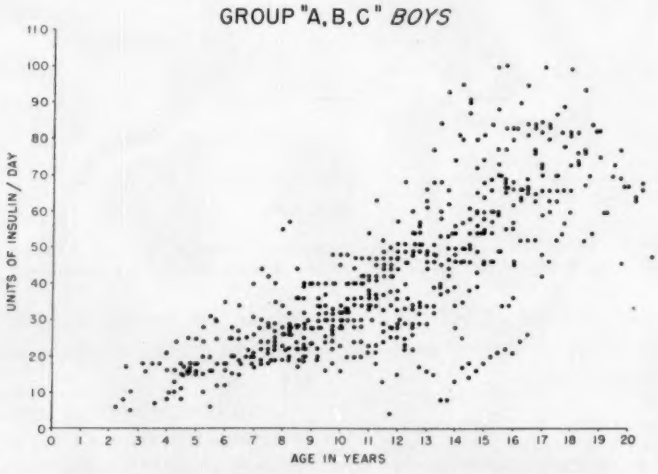


Chart 8.—The scattergram shows the varying insulin requirements of all age groups of boys (A, B, C) from infancy to adulthood.

and the increased need of insulin in relation to age and growth periods are revealed graphically in this distribution of insulin requirements for girls of given ages.

Chart 8, similarly to Chart 7, shows the insulin requirements of boys under good control for the combined groups, A, B, and of two boys and a girl from onset to last observation of this study. These curves demonstrate the interrelationship of insulin requirement and growth.

Chart 12 represents the average insulin requirements of yearly age groups of the girls from 3 to 20 years. Plotted also on this chart

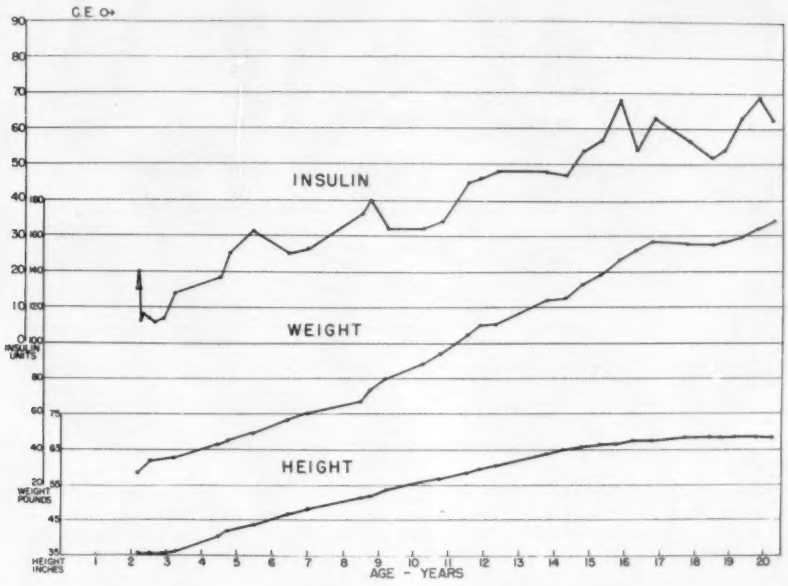


Chart 9.—C. E., boy, age of onset 2 years 2 months. Curves represent the growth in height and weight and the corresponding insulin requirements at varying ages during childhood and adolescence.

are the mean heights and weights for given ages of the study group in relation to the Iowa Growth Charts. Similarly, Chart 13 presents the average insulin requirements of the boys and shows the mean heights and weights for given ages of the study group in relation to the Iowa Growth Charts. The upper and lower curves of the insulin requirement are the mean +1 standard deviation and the mean —1 standard deviation for given ages, respectively.

The insulin requirement curves at the beginning of treatment decreased rapidly to a low intake for a short period after metabolic restoration (Charts 9, 10, and 11). After the sudden initial fall, the insulin requirement increases at a more rapid rate for another short period. During the period of lower insulin requirement, there is little or no growth in height, and during the period of increasing insulin requirement, an acceleration of linear growth.

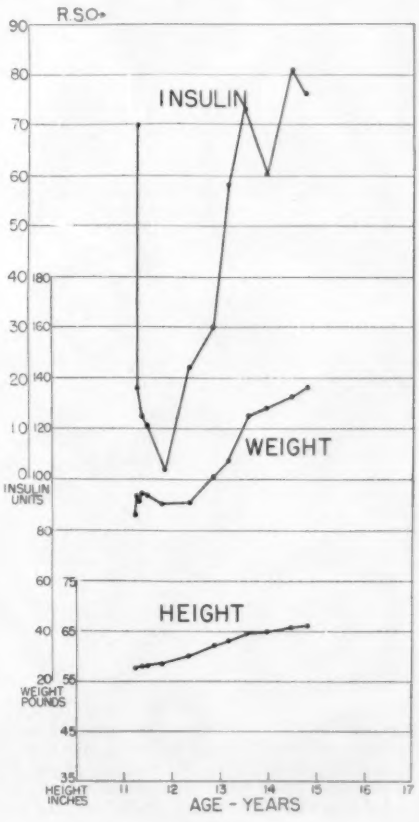


Chart 10.—R. S., boy, age of onset 11 years 2 months. Curves represent the growth in height and weight and the corresponding insulin requirements during the next four years.

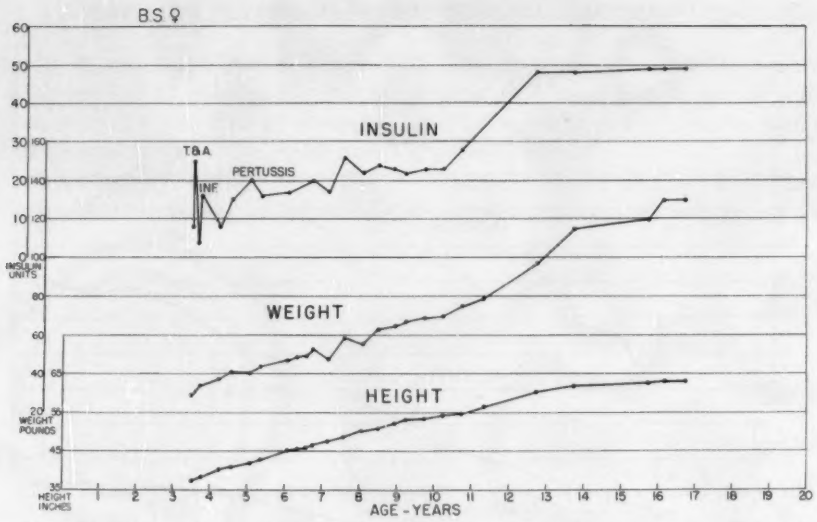


Chart 11.—B. S., girl, age of onset 3 years 4 months. Curves represent the growth in height and weight and the corresponding insulin requirements throughout childhood and adolescence. This child had repeated infections during early childhood, and they are indicated on the insulin dosage curve.

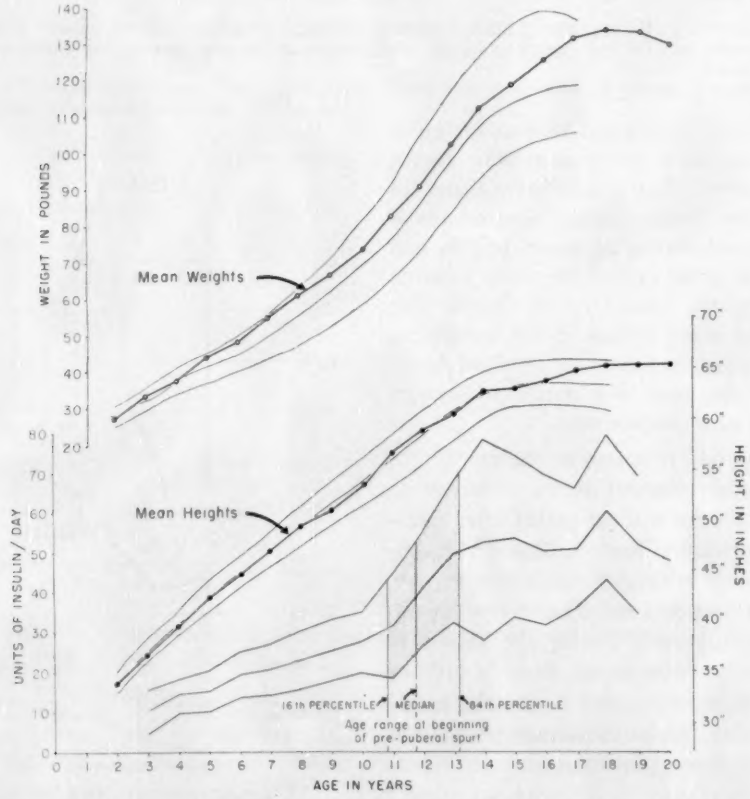


Chart 12.—Lower set of curves represents the mean insulin requirements of girls and a range of one standard deviation above and below the mean value. The other two sets of curves are the mean heights and weights of the group plotted in relation to the Iowa growth charts. In grouping these data, age was considered as the nearest year, for example, insulin values observed between 2½ years and 3 years 5 months were considered as the three-year requirement.

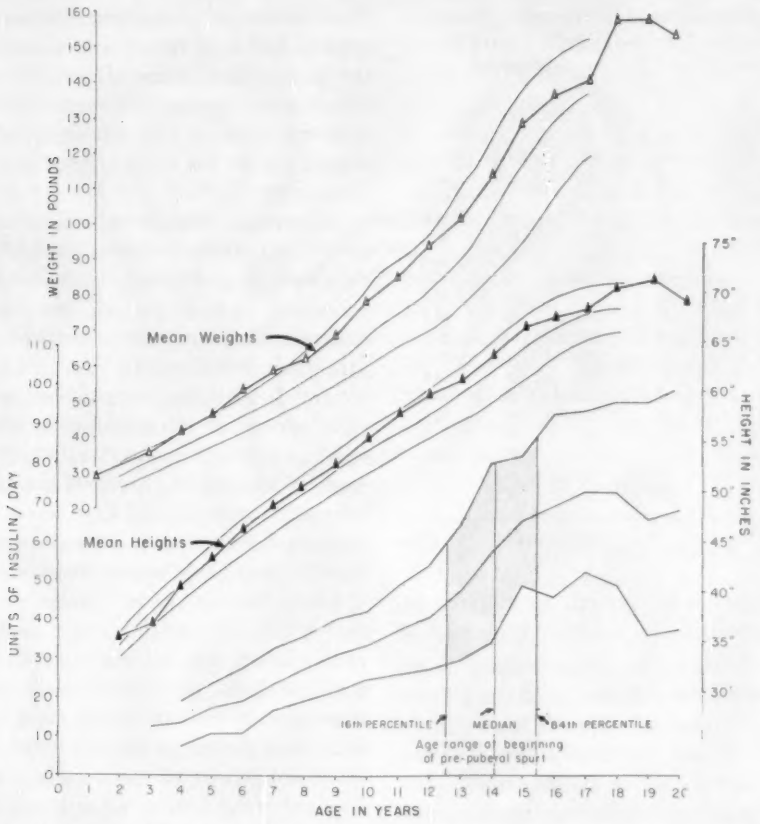


Chart 13.—Lower set of curves represents the mean insulin requirements of boys and a range of one standard deviation above and below the mean value. The other two sets of curves are the mean heights and weights of the group plotted in relation to the Iowa growth charts.

Up to 10 years, the growth patterns of the boys and girls are quite similar, in contrast to the great difference after 10 years as shown in Charts 12 and 13. The insulin requirements reflect the periods of similarity and differences in the sexes and clearly show a relationship of insulin requirement to growth. One notes the smoothness of the rise of insulin requirements in harmony with the fairly uniform growth in the midchildhood period. Chart 14 demonstrates that the boys' requirement is slightly greater in keeping with their somewhat greater body mass. The insulin requirement of girls increases rapidly after 10 years, concomitantly with their prepubescent growth spurt, and decreases after the period of rapid growth. The insulin requirement of boys increases rapidly after 12 years, concomitantly with

Chart 14.—The average insulin requirement curves of girls up to 10 years (from Chart 12) are superimposed on the average insulin requirement curves of boys (from Chart 13) to show the similarity of their pattern and range of insulin requirements during childhood.

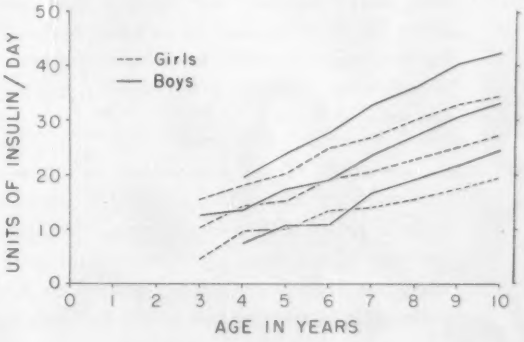


TABLE 2.—*Mean Calories per Pound of Weight of Twenty-Six Girls and Twenty-Two Boys with Diabetes Mellitus Maintained in Good Control*

Age, Yr.	No Observations *	Girls	No. Observations	Boys
2	2	37.2	2	40.0
3	4	35.0	4	34.9
4	10	35.7	10	33.0
5	14	32.6	12	32.7
6	18	32.7	14	33.2
7	20	32.9	16	32.9
8	24	32.5	22	32.8
9	26	30.4	24	30.8
10	28	29.0	26	29.5
11	34	26.6	28	27.7
12	37	24.4	28	26.3
13	39	22.3	28	24.5
14	36	20.2	28	23.6
15	30	18.2	28	22.5
16	28	17.5	28	21.5
17	26	16.4	24	20.6
18	18	15.3	22	20.3

* Not more than two observations per year for a subject were included in an age group.

their prepubescent growth spurt, and decreases gradually as adulthood is reached.

Table 2 shows the mean values of the calories per pound of weight of the 26 girls and 22 boys at yearly periods from 2 to 18 years. They are essentially the same up through age 10 years for boys and girls. From 2 to 3½ years, the calories per pound decrease rapidly, then level off until 8 years of age. From this age on through adolescence there is a steady decline. However, from age 10 to 18 years, the calories per pound of weight for girls decrease more rapidly than for boys.

The ratios of the daily insulin requirement to height, weight and surface area were computed for the boys at varying ages. The mean ratios for the different age groups were more constant for weight than for either height or surface area. Table 3 shows the mean and standard deviation of the ratio of insulin to weight for the boys in varying age groups.

COMMENT

This study of the exogenous insulin requirement per day of well-controlled juvenile diabetic patients showed a pattern of changing rate of increase which correlates with what has been observed for growth as revealed by measurements of height and weight. The correlation of insulin requirements and growth has been traced and shown (1) in the serial observations of patients and (2) in the comparison of the curves of mean insulin requirement and the mean height and weight curves for varying ages and periods of growth.

In retrospect, when we review and compare the curves of height, weight, insulin requirement, and caloric intake of the well-controlled diabetic patient, we discern the interrelationship of caloric and insulin needs and gains or failures to gain in height and weight. During the prepubescent and pubescent period, an acceleration in growth of height and weight, as well as other manifestations of maturation, are predictable for the normal boy or girl. We have found that this acceleration in growth is accompanied by a rapidly increasing insulin requirement.

For a majority of the patients, we noted that the insulin need for the size and age of the patient was low after initial regulation and metabolic restoration for a period of months, but then increased gradually until it reached the range found for the group of diabetic children of the same age and size. The amount of insulin required and the duration of the period of metabolic recovery depend on how long the state of hypoinsulinism and catabolism have persisted.[4] After stabilization is achieved, the insulin requirement

TABLE 3.—*Insulin per Pound of Weight of Boys with Diabetes Mellitus in Good Control in Relation to Age*

Age, Yr.	No. Observations	Mean	Standard Deviation
3	3	0.37	....
4	3	0.27	....
5	10	0.41	0.10
6	7	0.33	0.08
7	9	0.44	0.15
8	12	0.43	0.11
9	12	0.45	0.12
10	14	0.35	0.10
11	12	0.37	0.12
12	17	0.39	0.11
13	11	0.40	0.15
14	15	0.41	0.18
15	18	0.52	0.18
16	11	0.47	0.16
17	12	0.49	0.16
18	6	0.49	0.16
19	7	0.46	0.09
20	5	0.49	....

becomes fairly constant if the caloric intake is maintained at a relatively fixed amount. During this early period of treatment, the weight of the child increases rapidly, but height remains almost stationary. However, within a short period, the child's height begins to accelerate, and concurrently with this increase in growth rate, the insulin requirement increases and eventually attains in general almost a constant ratio with body weight (Table 3).

An evaluation of the daily insulin requirement, weight, and body build of an individual patient in good control in relationship to the range of values for insulin and growth of a group of well-controlled diabetic children gives some indication of the degree of hypoinsulinism. Extremely low values for a small percentage of our juvenile patients suggest the possibility of a different type of diabetes.

The findings of our study are in keeping with the data presented by Wrenshaw and Ritchie[5] on the amount of extractable insulin from pancreas of diabetic and nondiabetic children and the insulin requirements of adult patients who had to undergo total pancreatectomy, reported by Joslin, Root, and White[6].

A knowledge of insulin requirement provides useful information for clinical application as a guiding factor for maintaining good control and normal growth. For example, during the early months of treatment, the insulin and nutritional requirements change concurrently with marked changes in growth of weight and height, and that accounts for the difficulty in attaining and maintaining good control of the disease.[4] The rate of change is dependent on whether or not the disease has its onset in midchildhood or just prior to or concurrently with the prepubescent growth spurt, being accentuated during the period of rapid growth. However, after this period of changing body composition, a definite pattern is established. It is of some importance to know and to be able to predict the future requirement of a given child, to allay the fears on the part of the child and the parents who may be assuming that the disease is growing worse, as in the succeeding years he requires more insulin. The predictability of pubescent requirements, especially of those who are rapidly increasing their insulin dosage from low to high amounts, corresponding with their spurt of growth, will help immeasurably in the management of the diabetic patient during this difficult period. As adulthood is reached, it is equally important that the insulin dosage and caloric intake are adjusted to the person's adult requirement. This has been demonstrated well in the older girls of the group. At this period, the adolescent girl will become obese if her insulin and caloric intake are not adjusted to the lower requirements of an adult.

SUMMARY

A study of the serial insulin requirements and growth of 48 well-controlled juvenile diabetic patients revealed a relationship between the changing insulin requirement and the growth of girls and boys. The age of onset of the disease did not influence materially the insulin requirement. A knowledge of the insulin requirements and growth at varying age periods of well-controlled diabetic patients helps in the evaluation of the degree of hypoinsulinism of individual patients and in the prediction of insulin and nutritional requirements for maintaining good control of the disease and a normal growth pattern.

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PSYCHOLOGIC ASPECTS OF MANAGEMENT OF CHILDREN WITH MALIGNANT DISEASES

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THE MANAGEMENT of patients at any age with malignant disease is a trying experience for the physician as well as for the patient and his family. When such disorders arise in childhood special problems are presented. Therefore, it occurred to us that observations made during our management of 48 children with leukemia [1] and many children with other malignant lesions would be of value to others faced with similar clinical problems. Although many patients in this study were hospitalized for metabolic observations of the effect of folic acid antagonists administered experimentally, we do not imply that prolonged hospitalization is necessary or desirable. Because of the tragic nature of this group of diseases, it is not a simple matter to undertake a carefully controlled study of emotional reactions of either patient or family; it is hoped that some relatively innocuous methods of measurement may be developed and applied in the future. This report, however, is based solely upon our clinical impressions.

As death from infectious, nutritional, and metabolic disturbances decreases in incidence, malignant disorders assume relatively greater significance in the practice of the pediatrician. One author has indicated that 3% of surgical patients admitted to a children's hospital had malignant disease and stated that "it is probable that cancer in childhood will become an even more frequent cause of hospital admissions." [2] If leukemia, lymphoblastomas, and other nonsurgical malignant disorders are added, hospital practice is increasingly concerned with the management of children with potentially fatal diseases and their families. In this light it is striking to note that so little has been written concerning this aspect of pediatric practice, although Alvarez has recently presented an interesting discussion of this and similar problems in adults.[3]

At the outset it must be stated that these comments will necessarily be general. In dealing with matters as complex as the human personality, the spectrum of reactions is so broad as to minimize the significance of generalizations. Our observations are not intended as a substitute for study and evaluation of each patient and family. Intelligent management is dependent upon some understanding of the background of each individual, as it is in every situation involving the study of human behavior. Indeed, with a knowledge of parental personalities, the experienced observer can make reasonably accurate predictions of what may be expected under emotional stress.

The diagnosis of malignancy necessarily brings with it one of the most deep-seated of all anxieties, that of separation from a loved one. This has deeper meaning than most separation anxieties which parents and children face because of the finality of separation which comes with death. The psychological depth of this reaction indicates the importance of the physician's precision in diagnosis; without an accurate diagnosis it is cruel to arouse anxieties unnecessarily, although publicity and cancer education have made people increasingly aware of and sometimes needlessly anxious concerning the possibility of this diagnosis. This emphasizes the importance of ready availability of diagnostic consultation services in which there is specific competence in neoplasms of children by physicians, since it is impossible for individual physicians to

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encompass personally all the subleties of malignant diseases of childhood.* The relative significance of these diseases renders it imperative that complete frankness—again, without arousing undue anxieties—obtain in the relationship between physician and parents. Procrastination in diagnosis or imparting inaccurate knowledge concerning the diagnosis often renders future relationships—and indeed the relationship with all physicians—more difficult. Thus, promptness and skill in diagnosis of the physical disorder may have far-reaching effects psychologically in management.

For purposes of presentation, the observations relevant to the patient, parents, physicians, and other personnel will be discussed separately. Obviously this division is arbitrary, since interaction among these persons is continuous. This interaction will be brought into the discussion as is feasible.

THE CHILD

The reaction of the child to malignant disease is dependent to a considerable extent upon the age at which the disease develops, since there are different levels of understanding at different ages. The type of lesion probably is also a significant factor in how the child views the disease. Changes in energy for physical activity (as in leukemia) or obvious physical abnormality (as in sarcoma about the neck or face) must influence how the child views himself and his relationship to others.

Prior to the establishment of the diagnosis in leukemia it is not uncommon for a child to be lacking in energy sufficient to carry on his usual daily activity. Since the cause for this change in behavior is not recognized, he may be subjected to criticism and to punitive action by siblings or parents (with later feelings of guilt on their part) because he does not respond in his usual manner and assume his usual responsibilities. Or he may be subjected to the comments of other children concerning obvious bodily deformity. Or, if surgery is being considered, the realistic loss of a part of the body and its symbolic meaning may be productive of great anxiety. Psychiatric consultation may be very helpful in identifying the nature of the child's problem.

In spite of the above factors, children observed by us rarely manifested an overt concern about death. Even among adolescents, who intellectually may know much about cancer, the question concerning diagnosis and possibility of death usually was not raised as it often is by the adult patient. Our suspicion is that this does not reflect an awareness but rather represents an attempt at repression psychologically of the anxiety concerning death. For the occasional child who asked about his diagnosis, a simple descriptive statement was given without provision of a diagnostic label.

In general, children seem to have reacted with an air of passive acceptance and resignation. Associated with this there often seemed to be an atmosphere of melancholia. Some of this may, of course, be the projection of the staff and parents rather than the child's feelings. Since the child's physical energy diminishes so significantly, it may be that much of the child's lack of emotional as well as physical response is due more to this factor than to psychological awareness of the diagnosis.

During diagnostic and therapeutic procedures we have noted a tendency to greater passivity to the procedures than is present in normal children. Whether this is due in part to resignation or to the lack of physical energy is not known at present. With the progress of the disease the passivity tends to become marked; it is not uncommon to observe children with extensive lesions and hemorrhage maintained entirely by parenteral fluid therapy, without any significant protestations concerning its discomfort.

PARENTS

One of the physician's most important roles in the management of the child with a malignant disease is the help which he may provide

* If facilities are not available locally for physicians, the Tumor Registry of the American Academy of Pediatrics, Dr. Harold Dargeon, Chairman, 112 E. 74th St., New York 21, provides an excellent consultation service.

to the parents in their integration of a tragic event into their life experiences. Perhaps at no other time does the previous integration of the personality and of the family unit reflect itself so clearly as it does during the severe emotional stress associated with mourning. The physician should realize that the withdrawal, feelings of unworthiness, and preoccupation with thoughts of earlier experiences with the loved one for a period of time (usually several weeks) is part of the mourning process in the normal person. When these manifestations are extreme, it may be suspected that parental psychopathology is present and that psychiatric help may be necessary. Ordinarily, this period serves as one in which a reintegration of feelings takes place and a gradual redirection of energies externally occurs. It should not be the physician's goal to minimize or to abort what is a physiologic emotional experience. There is perhaps too great a tendency to attempt to spare people from this experience in our culture, which probably is not desirable. Many cultural practices concerning paying respects to the bereaved, centering around the provision of help in ways unique to each cultural group, have respected the emotional implications of mourning.

The physician can help in relieving the suffering of parents who are mourning if he has insight into the feelings which they are experiencing. These feelings initially center about the anxieties concerning parental responsibilities for the development of the illness. These are usually manifested as feelings of guilt. When given an adequate opportunity to express their anxieties, almost all parents will ask questions ranging from "whether it would have been different if we had called the doctor earlier" to "do you think my spanking him may have brought this on?" The deep emotional basis of these questions is revealed by the fact that they arise in spite of the fact that intellectually most of these parents know better. The ventilation of these anxieties has therapeutic value; reassurance within the limitations of our knowledge of etiology is helpful. We refer to this as the physician's "sharing" of anxiety, which has the effect of reducing its intensity. It is important, however, to permit the parents to verbalize sufficiently to determine the specific anxieties. A case in point was the futile attempt of a house staff member to reduce the anxiety of a father whose son was admitted with a prior diagnosis of acute leukemia. The young physician's own anxiety led him to extended explanations of the disease, the lack of a known etiology, etc., with no results. A senior staff member observing the situation casually engaged the father in conversation and within a few minutes learned that his anxiety centered about whether he was going to be a recipient of "charity" against his will. When assured there was no rule against his paying the hospital bill he was considerably more comfortable. He had already made a reasonably good adaptation to the child's illness.

A second major concern of parents, as previously indicated, centers about separation and its irreversibility. The rearing of children represents a series of steps in separation from the birth process to the grave. When death comes prematurely, the blow is considerably more potent. Aspirations and plans which have developed over the years are shattered, for what parent has not dreamed of what a child will grow up to be? The physician is therefore called upon to help parents deal with the feeling of emptiness which characterizes the separation from a loved one during mourning.

Malignant disease is not usually associated with sudden death. The physician, therefore, has time in his favor in helping parents through the severity of this separating experience. Although depression or marked anxiety may accompany the initial impact of the diagnosis, the period of illness prior to death may be one of reintegration of parental feelings in which the mourning process takes place substantially prior to the death of the child. This may occur with any prolonged fatal illness and is observed with adult patients and their families too. The fact that the separation itself occurs over a period of time tends to facilitate this. Our experience

indicates that parents are often enabled to traverse this difficult emotional experience with much less difficulty in this way. This is in contrast to the feelings on the part of friends or some physicians who, reflecting their own anxieties, feel that it is "better to get it over quickly."

We have found that the involvement of parents in the physical care of the child is extremely important in facilitating parental adaptation. First, this permits the parents to have a feeling that they personally have done everything possible for the child; second, feelings of guilt are somewhat relieved by the expenditure of personal effort in the care of the child; third, in retrospect parents are very grateful for having had the opportunity to spend as much time with the child as was possible, and fourth, an opportunity to observe and to participate in measures directed to relieve pain and discomfort as much as possible is comforting to parents. Except for the unusual instance of very disturbed parents, in whom there were preexisting problems in the parent-child relationships, there was a real feeling of involvement in hospital care of the child. The parents were given an opportunity to participate realistically in the physical care of the child; they were gainfully occupied in routines of bed making, feeding, play activities, story reading, etc. Thus, parents became integrated into the ward program and communication with ward personnel was enhanced thereby. This tended to effect some interidentification with parents sharing to some extent the problems of nurses, doctors, and other staff, while the staff in turn shared parental problems.

An observation which seemed interesting to us and which further illustrates the advantage of parental participation in physical care relates to the development of parental capacities to care for and to give of themselves to other children. Usually the initial period on the ward was one of involvement solely with their child. After a variable period, a desire to help as effectively as they could with the care of other children developed. In many instances it was our feeling that the manifestation of this capacity to give to other children marked a turning point in parental adjustment which reflected acceptance of the child's illness and ultimate death, with the conversion of energy which had previously gone into a form of mourning to more constructive endeavors. This observation is in accord with that noted by Freud [4] many years ago as marking the end of mourning.

Many of these parents ultimately continued their contact with ward personnel and desired to be of service; others found emotional outlets in the provision of gifts to the ward or to children on the ward. In a more organized way a sizable group of these parents have become active in the promotion of research in hematologic and malignant disorders. These activities were never encouraged by the staff in any formal way, but it was interesting that the contacts of parents with others sharing the same problem permitted the development of what seemed to be an organized yet socially and emotionally desirable form of activity.

Because of the considerable anxiety which parents of a child with a malignant disorder faced, we believed it undesirable to add to this anxiety by leaving decisions concerning treatment to them. We are aware of the fact that it is not uncommon for physicians to leave a decision to parents as to whether an attempt at treatment is to be instituted, with potential prolongation of life. We believe that the feelings of guilt which every parent has to some extent are too great to permit the intensification which may occur when the parent is placed in a position of making a decision which may or may not alter the course of the disease. We therefore have assumed the responsibility generally of offering an opinion to parents concerning what we believe to be desirable in management. It has been only in the rare instance when the parent has in a very positive way indicated "If he has to go, we want him to go as fast as possible" that we have not assumed complete responsibility for medical management.

Although parental participation in the care of the sick child is desirable, this should not be at the expense of the emotional and physical well-being of the remainder of the family.

Siblings may be particularly disturbed during this period and may require considerable parental support. It is helpful therefore if the physician encourages parents to divide their time among the various members of the family as the situation warrants. This provides a greater sense of sharing of the problem by all members of the family; greater integration of the family unit usually develops as a consequence.

PHYSICIANS

The physician's relationship to the patient and family is considerably influenced by his developmental background. As the family experiences the emotional problems of separation, the physician, by understanding and empathy, also experiences significant reactions. These are especially difficult for the young physician in many instances.

When the disease process is chronic, an opportunity for identification rather than empathy with the patient and family may develop. This may consume a considerable portion of the physician's energy—either by driving him to extreme (and often absurd) lengths to "do something" or by influencing him to "do nothing so that the end will come soon." The response of each person varies in relation to many factors in the patient's and the physician's backgrounds. As an example, overinvolvement with one patient was observed in an otherwise fairly objective physician; it was noted that this physician unconsciously identified the patient with his son, who happened to have the same given name and was of the same age. As a consequence, he went to extremes to "leave no stone unturned" and in the process tended to subject the child to many more therapeutic procedures than were indicated. It probably need not be emphasized that such emotional relationships, aside from being difficult for the physicians, interfere significantly with objectivity in providing medical care.

Senior staff members should also be aware of the dissipation of emotional energy which may occur when a relatively youthful house staff has several children with malignant disease under constant care. Without their conscious awareness, we found that house staff members tended to find many reasons (beyond the reality of physical work involved) for not admitting additional patients with leukemia if they had as many as three such patients under their care. In effect, they were indicating to the senior staff their limits of tolerance for such emotional stress. It was also not uncommon for house staff members to experience a considerable feeling of relief on leaving the services which was disproportionate to its physical and intellectual demands and which seemed greater than that observed prior to our study, when patients with malignant disorders were not so numerous on the ward.

It has been striking to observe the respect and gratitude which parents have for the physician in the face of his inability to provide specific therapy. The opportunity which the physician has to share parental anxieties, his permitting some dependency in a situation which makes real demands for maturity, and his provision of realistic help and interest make for a relationship which is appreciated deeply. This is not unique to the management of children with neoplastic disease; we have also observed it in parents of children with muscular dystrophy, when the physician is often embarrassed by the gratitude of parents in the face of his helplessness in combating the progress of the disease.

OTHER STAFF

Much of the previous discussion applies to other hospital personnel. Nurses play a particularly important role, since they provide much of the physical, as well as psychologic, care of patients. That they should be selected on the basis of their real interest in and love for work with children is an obvious statement, which is not always respected. Patients and parents readily detect feeling tones even if these are not verbalized, and serious doubts concerning adequacy of care may enter the parents' minds when members of the nursing staff are not deeply interested in children.

The acceptance by the nursing staff of the continuous presence of parents was not as difficult as we believed it might be initially. Ordinarily many nurses believe it more effi-

cient to perform all the work involved in patient care without "interference" from outsiders. The severe illness and many needs of the patients, however, convinced them that the children usually received better care when parents were present. Genuine acceptance of parents and considerable sensitivity to and understanding of their problems were observed in the nursing staff after the program was in progress for several months. Hospital administrators were also won over to this policy, even though no adequate facilities were available for parents. Improvisation by the nursing staff to care for parental needs developed spontaneously. The effectiveness of this program with nurses, dietitians, occupational therapists, teachers, aides, and others, we believe, is attributable largely to the example set by the more senior members of the pediatric staff and the identification which the remainder of the staff achieved with them.

SUMMARY

Observations, experiences, and suggestions for management of children with malignant diseases and their families based on a study of 48 children with leukemia and children with other neoplastic diseases are presented. Suggestions by which the physician may help such children and families through the trying emotional experiences associated with these diseases and their fatal outcome are discussed.

The house staff and nurses (under the direction of Mrs. Helen Mack, R.N.) and other personnel of the pediatric department of the Research and Educational Hospital of the University of Illinois College of Medicine participated in the care of patients who were the subjects of this study.

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RESPIRATION AND SPEECH IN THE CEREBRAL PALSIED CHILD

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INFANTILE cerebral palsy affects the brain centers of respiratory regulation, as well as the pharyngeal and laryngeal muscles of respiration, speech, and deglutition. Proper respiration control is prerequisite to proper speech; children with poor respiration control have, for the most part, poor speech. This is irrespective of the level of intelligence if speech performance ability is considered rather than cerebration and thought content.

Infantile cerebral palsy is a unique syndrome in that it involves so many peripheral structures and body functions, all consequent to central brain damage. The etiological factors are cataclysmic and of short duration, e. g., cerebral anoxia, trauma (usually hemorrhage), brain infection, and so on. Therefore, the damage is not progressive. However, since it occurs mainly prior to or during birth, at the outset of the person's life, the condition dynamically affects the entire physical, mental, and functional development of the afflicted person. Thus speech, which is a complex function dependent upon physical, mental, emotional, and social factors, is frequently affected by cerebral palsy.

The physiology and neuroanatomy of respiration may be reviewed in any standard texts on the respective subjects (Wright,[1] Ranson [2]). However, certain pertinent points bear noting. Respiration is an automatic reflex act of considerable complexity. Anatomically, it involves the abdominal muscles, the diaphragm, the chest cage muscles, and the accessory (chiefly neck) muscles of respiration. Afferent stimuli arise in the alveoli of the lungs and are carried by the vagi to the respiratory centers in the medulla. These impulses signal the degree of lung expansion to the brain-stem centers, which then check inspiration and initiate expiration (Hering-Breuer reflex).[1] Other afferent stimuli, such as nose, throat, and tracheobronchial mucosal irritations, cause alterations in respiratory rhythm like sneezing and coughing. The $O_2 : CO_2$ ratio in the blood is one of the most important stimuli to act in a chemical way upon the respiratory centers of the brain and upon the carotid and aortic bodies. Laurens' review of the relevant neuroanatomy [3] places the inspiratory and expiratory centers in the medulla at the level of the vagus nuclei and near the nerve nuclei which innervate the muscles of the glottis, facial muscles, and diaphragm. The two respiratory centers receive afferent fibers from the pneumotaxic center near the hypothalamus and from the solitary nuclei. The latter are influenced by impulses from the carotid and aortic body chemoreceptors and from the stretch receptors in the lungs.

Pohl [4] notes that breathing disturbances are common in cerebral palsy owing to impairment of control of the respiratory muscles. The thorax and abdomen frequently are not synchronized in their movements, causing breathing to be arrhythmic and speech to be spasmodic, irregular, and weak in volume.

Brock [5] declares that since the automatic rate of breathing can be altered by voluntary acceleration or slowing, it is obvious that there is a central control still higher than the brain stem. The ease with which laughter or weeping disrupt the normal respiratory sequence is still further evidence of higher control. Thus, it may be postulated that irregularities in respiration in cerebral palsy may be due to direct central factors as well as to peripheral difficulties with the various muscles of respiration.

From the Cerebral Palsy Unit, Public School 118, Queens, Bureau for Handicapped Children, Department of Health, New York.

The hypothalamus is the highest vegetative center in the brain. It affects the medullary respiratory centers through the pneumotaxic center and so plays a significant role in the regulation of respiration and respiratory rhythm. To a variable extent, voluntary higher cortical control may be exercised over the hypothalamus and lower centers in learning to regulate respiration. Because of its contiguity to the basal ganglia, which are the areas more or less seriously damaged in the athetotic person, the hypothalamus may also be affected by the original traumatic factors—frequently cerebral anoxia for a prolonged period at the time of birth. In the spastic child, the damage is more in the cortex than in the basal ganglia. Therefore, here there is less likely to be damage to the central respiratory regulating mechanism.

Respiration is one of the most important parts of speech, since it produces the air currents for phonation as well as for voiceless sounds. It is also an essential factor in proper speech phrasing. Ability to speak also requires certain other factors. There must be afferent pathways for sensory impressions: auditory, visual, and tactile. The latter two are especially important where hearing impairment exists. A certain degree of intactness of cerebral function must be present in the form of operating sensory, thought and association, and speech areas. The areas between the first temporal and the opercular part of the third frontal gyrus are concerned with speech, but a further subdivision into motor and sensory speech centers has never proved feasible. The speech centers are contiguous with the reading (visual speech) and writing centers. A certain level of intelligence is required also. Efferent neural pathways and functional effector organs, such as the muscles of the mouth, throat, chest, and abdomen, are the remaining members of the speech faculty.

Speech difficulties encompass a wide range, from faulty pronunciation and enunciation to complete mutism. The factors may be prenatal or congenital, paranatal, or postnatal. The causes may be physical or psychogenic.

The commonest causes of dysarthria in the cerebral palsied child are the brain injuries occurring during birth which produce faulty control of the muscles of the face, mouth, tongue, and larynx that are concerned with speech. Some failures in speaking where peripheral apparatus is relatively intact may be the result of a severe degree of mental deficiency accompanying the cerebral palsy. Others may represent a primary aphasia consequent to damage to or maldevelopment of the cortical speech area. Still others may ensue from deafness of varying degrees with failure to appreciate sound and pitch appropriately. Psychogenic factors of insecurity, dependency, anxiety, and so on can affect the cerebral palsied child as well as any other child and produce infantile, retarded, or otherwise defective speech. Finally, improper, irregular respiratory functioning and control may be manifested in poor phonation resulting in weak volume, tonic halting speech, and inspiratory instead of expiratory speech, to mention but a few defects.

MATERIAL AND METHODS

Observation of a group of 27 cerebral palsied children, ranging from 2½ to 12 years of age, of whom 13 were spastic, 12 athetoid, and 2 ataxic, revealed that in general speech was better in the spastics than in the athetoids, with respect to volume, phonation, and general control. Furthermore, respiration was better in the spastics in respect to regularity of rate and rhythm, depth, and volume. Respiratory rate was counted, and the quality of the rate, rhythm, and depth was estimated by listening to each child's breathing in a sitting position for two consecutive minutes, with a stethescope placed over one of the upper main bronchi anteriorly.

Of the 13 spastics, all had fair or good speech. Of the 12 athetoids, 3 had fair or good speech and 9 had poor or no speech. Two of the latter had inspiratory speech. One ataxic child had good speech, and the other had none. In the series of 27 children,

of the 18 with good or fair speech 15 had regular rate and rhythm of respiration (1 had regular rate but slightly irregular rhythm) and 3 had irregular rate and rhythm. Of the nine with poor or no speech, two had regular rate and rhythm and seven had irregular rate and rhythm. Irregularity or shallowness of depth of respiration was associated with irregular rate and rhythm. Among the children with regular rate and rhythm, there were only two with shallow depth and none with irregular depth of respiration.

An attempt was made to allow for mouth and tongue difficulties in evaluating quality of speech in order to equate the two categories of spastic and athetoid. Thus, quality of speech was judged on the basis of ability to produce smooth vocal and aspirate sounds in the larynx and to phrase speech, rather than on the basis of facility in modifying the sounds by means of lips, tongue, and cheek control. The terms good, fair, and poor used in relation to quality of speech were subjective evaluations of the above-defined abilities agreed upon by the speech therapist and me.

There seemed to be no direct correlation in this group of children between speech achievement and intelligence quotient.

TABLE 1.—*Relationship of Diagnosis, Speech Quality, and Respiratory Efficiency*

Patient	Name	Age, Yr.	Diagnosis *	Speech Quality	Resp. Rate/ Min.	Rate	Rhythm	Depth	I.Q.
1	S. Z.	5½	Sp. para.	Good	28	Reg.	Reg.	Reg.	102
2	J. O.	7	Sp. para.	Good	24	Reg.	Reg.	Reg.	90
3	J. Sp.	5¾	Sp. quad.	Good	18	Reg.	Reg.	Reg.	103
4	J. Se.	9	Sp. quad.	Good	16	Reg.	Reg.	Reg.	90
5	J. Gr.	5	Sp. para.	Good	24	Reg.	Reg.	Reg.	121
6	K. B.	6½	Sp. para.	Good	17	Reg.	Reg.	Reg.	57
7	G. L.	4¾	Sp. para.	Good	29	Reg.	Reg.	Reg.	84
8	C. S.	5	Sp. quad.	Good	24	Reg.	Reg.	Reg. shallow	144
9	B. N.	7	Sp. para.	Good	18	Irreg.	Irreg.	Irreg. shallow	66
10	W. C.	5	Sp. quad.	Fair	22	Reg.	Reg.	Reg.	73
11	N. D.	10½	Sp. quad.	Fair	20	Reg.	Reg.	Reg.	97
12	S. LoC.	6	Sp. quad.	Fair	28	Reg.	Reg.	Reg.	85
13	L. L.	7¾	Sp. quad.	Fair	27	Reg.	Irreg.	Irreg.	80
14	M. G.	2½	Athetoid	Good	20	Reg.	Reg.	Reg.	WAA †
15	S. Sl.	12	Athetoid	Good	22	Reg.	Reg.	Reg.	114
16	F. F.	11	Athetoid	Fair	16	Reg.	Reg.	Reg. shallow	89
17	B. A.	6½	Athetoid	Poor	23	Reg.	Reg.	Reg.	84
18	M. C.	12	Athetoid	Poor	20	Reg.	Reg.	Reg.	74
19	C. H.	9	Athetoid	Poor	26	Irreg.	Irreg.	Irreg.	67
20	S. Sl.	9½	Athetoid	Poor	16	Irreg.	Irreg.	Irreg.	96
21	J. M.	6½	Athetoid	Poor	31	Irreg.	Irreg.	Irreg.	121
22	F. Y.	5	Athetoid	Poor ‡	27	Irreg.	Irreg.	Irreg.	97
23	N. G.	5¾	Athetoid	Poor ‡	21	Irreg.	Irreg.	Irreg.	98
24	P. McG.	11	Athetoid	None	26	Irreg.	Irreg.	Irreg. shallow	PAA §
25	J. Ga.	7	Athetoid	None ‖	24	Reg.	Reg.	Reg.	63
26	T. G.	7	Ataxic	Good	21	Reg.	Reg.	Reg.	81
27	S. K.	7¾	Ataxic	None	24	Irreg.	Irreg.	Irreg.	WAA †

* Sp. para. indicates spastic paraplegia; Sp. quad., spastic quadriplegia.
† Well above average.
‡ Inspiratory speech.
§ Probably above average.
‖ Probably aphasic.

These observations are summarized in Table 1. All the children were attending the Cerebral Palsy Unit at Public School 118, Queens, and were unselected, except No. 14, who was a private patient of preschool age.

These statistics show an unmistakable difference in the respiratory control and directly correlated speech quality between the spastic and the athetoid children, with the former much better than the latter. There

is also a proved direct relationship between the regularity of rate, rhythm and depth of respiration, and quality of speech. This is clearly in line with the hypothesis stated above concerning the hypothalamic influence on respiration and, therefore, speech.

Respiration training must have a twofold purpose: the proper usage of the "bellows apparatus"—the muscles of abdomen, diaphragm, and chest—and the increase in pulmonary tidal volume and vital capacity. The end-results of these achievements should be improved speech and, theoretically, increased resistance to respiratory infections because of greater and more thorough pulmonary air exchange. Cerebral palsied children do have a greater susceptibility to respiratory diseases because of shallow irregular respirations and diminished tussive effort.

TABLE 2.—*Average Percentages of Pupils Attending School*

	Cerebral Palsy Unit	Kindergarten	School as a Whole
November, 1952	89.5	79.5	91.2
December, 1952	78.5	77.5	89.0
January, 1953	73.5	66.8	87.2

A rough attempt at statistical support of this postulate was made by comparing percentages of attendance for the school as a whole, the kindergarten group, and the Cerebral Palsy Unit for November and December, 1952, and January, 1953, months when respiratory disease incidence is high (Table 2).

From general experience, it was assumed that most absences were caused by respiratory infections. Kindergarten children are generally kept home for slight reasons, while the cerebral palsied children, who have motor transportation, are sent to school more readily. Yet, the cerebral palsy group, ranging from 5 to 12 years of age like the total school population, showed an appreciably lower attendance record than the school as a whole, though somewhat higher than the kindergarten group, which averaged about 5 years of age. The difference of 10.5% for December and 13.7% for January between the attendance rates of the Cerebral Palsy Unit and of the school as a whole may be regarded as statistically significant. It must be emphasized that these are raw figures, presented to indicate a consistent difference rather than as a careful study in themselves.

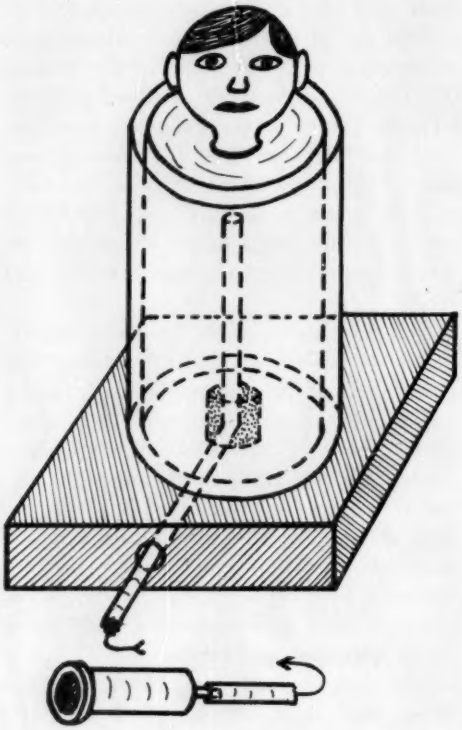
The speech therapist is concerned with developing and improving the afflicted child's use of the muscles of lips, cheeks, tongue, palate, pharynx, and larynx. This therapist deals with the modulation and control of expired air to produce speech sounds and, incidentally, with the child's ability to control drooling by proper swallowing in an efficient relation to breathing and speaking. Such methods are used as blowing out matches, blowing balloons, bubbles, harmonicas, and feathers, and licking peanut butter off lips, teeth, and spoons. There are also special exercises devised for palate and vocal cords.

A further technique of speech therapy is the development of regular breathing rate and rhythm in accompaniment to a ticking metronome or recitation and singing of simple rhymes. This method is particularly successful in the athetoid. It utilizes the voluntary cerebral control over the hypothalamic centers, which, in turn, regulate the medullary centers of respiration. This represents a practical application of the neurophysiology and neuroanatomy of respiration, which were discussed earlier.

Abodminal and chest muscle training exercises have been well described by Pohl [4] and others. The child practices breathing in supine, sitting, and standing positions, with the physical therapist's hands on the child's rib cage. The child also is allowed to feel the therapist's respiratory movements by placing his hands on the therapist's ribs.

I have introduced a few additions to these standard techniques which have been found helpful. With the child lying supine, extending his arms above his head lifts up the rib cage and increases the respiratory excursions considerably. Even vocal volume may be greater and speech louder and more sustained in this position. Another useful measure is to place a flat sandbag on the supine

child's abdomen. A 1-, 2-, or even 3-lb. bag may be used, depending upon the child's size and comfort and the efficiency of the results. This immediately increases rib exursions by impeding abdominal wall and visceral movements and larger diaphragmatic motion. It is not intended to enforce immobilization of the abdomen and diaphragm but merely to divert more of the work of respiration to the thorax. Both of these measures tend to improve thoracic breathing and regularity of the rate and rhythm of respiration.



Blowing apparatus for respiration training.

In May, 1952, I created and put into use in the Cerebral Palsy Unit at Public School 118, Queens, a device for respiration training based upon the principle of the water spirometer. The apparatus, which is still in use, consists of a round 10-oz. can inverted inside a round 14-oz. can, which is almost filled with water. The outer can is mounted on a wood block and pierced in the center of the bottom. A plastic tube inserted through a rubber stopper rises perpendicularly to a height just above the water level. The plastic tube is connected to a rubber tube which traverses a tunnel in the block. A detachable and sterilizable mouthpiece attaches to the rubber tube. The mouthpiece is flanged for easy gripping between the teeth and lips. The barrel of an ordinary 2 cc. hypodermic syringe was found to be satisfactory, but a similar metal tube is preferable. A doll's head made of paper or celluloid is cemented to the inverted inner can. A light cloth dress attaches at the doll's neck and covers the entire outer can. As the child blows into the tube, the inner can rises and makes the doll "grow tall," as a motivation for the blowing exercise. When the mouthpiece is detached, the air escapes easily and the inner can settles down again (Figure).

The apparatus has been used by the speech therapist in conjunction with speech training. Records of achievement have been kept for each child, i. e., the number of blows required to raise the doll to its maximum height. Many of the children showed some improvement, with fewer blows required, and a few showed marked improvement over a period of time. The achievement of raising the doll in fewer and fewer blows represented improving functioning and coordination of lips, cheeks, and tongue, along with the chest and abdominal muscles. There was a parallel, though not necessarily commensurate, improvement in speech volume and often in enunciation.

SUMMARY AND CONCLUSIONS

Proper respiratory function and control are of great importance for proper speech.

Cerebral palsy may frequently affect speech because of damage to central areas concerned with respiration and speech, as well as peripheral muscle involvement.

Athetoid children have poorer respiration control and, therefore, poorer quality of speech than do spastic children, largely because the former have damage to the hypothalamic respiratory regulating centers and the latter usually do not.

Respiration training should have a twofold purpose: first, the proper usage of the

"bellows apparatus" (muscles of abdomen, diaphragm, and chest), and second, the increase of pumonary tidal volume and vital capacity.

Cerebral palsied children may have a somewhat greater predilection for respiratory infections because of poor pulmonary dynamics.

Improvement in respiratory function produces some improvement in speech, although not necessarily commensurate, and, theoretically, increased resistance to respiratory infections.

Speech therapy is largely concerned with training of lips, cheeks, tongue, palate, and vocal bands.

Physical therapy helps respiration and, therefore, speech, mainly by the training of chest, abdominal, and diaphragmatic muscles.

Some additional methods of respiration training have been introduced, such as the use of the supine position with arms extended over the head and the placing of a sandbag on the abdomen.

A blowing apparatus is described which improves coordination of respiratory muscles and mouth and which helps to increase pulmonary volume and respiratory air exchange.

The speech therapists and the physical therapists of the Cerebral Palsy Unit of Public School 118, Queens, helped in this study.

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Case Reports

LYMPHOSARCOMA OF THE INTESTINE

Report of a Case in a Ten-Year-Old Child with a Seven-Year Cure

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LYMPHOSARCOMA of the intestinal tract in children is a rare disease, and rarer still are reports of its cure. The diagnosis is not readily made preoperatively, nor is the clinical picture often characteristic. Systemic spread occurs sooner or later in the neglected patient, and whatever the treatment, whether surgical or radiologic, there is little chance of cure.[1] Most authors on this subject believe that a combination of radical surgical extirpation of the involved bowel and mesentery followed by heavy roentgen therapy to abdominal and mediastinal areas offers the optimal chance of survival.[2]

Because of the rarity of this condition in children and the very low percentage of cures, this case with an apparent seven-year cure following surgical resection and roentgen therapy is presented.

REPORT OF CASE

C. S., a 10-year-old boy, was admitted to the service of one of us (H. M. E.) on April 26, 1947, with the chief complaint of abdominal pain for several hours prior to admission, which had entirely subsided at the time of admission to the hospital. His first attack of pain occurred one year prior to admission, following a heavy meal. He had no further symptoms until nine days previously, when he had two episodes of abdominal pain and vomited once. His temperature was normal on admission. The past history disclosed measles, chicken pox, and tonsillectomy.

From the Pediatric Service of Lebanon Hospital; Attending Pediatrician (Dr. Eisenoff) and Adjunct, Pediatrics (Dr. Klinger).

Physical examination revealed a well-developed and well-nourished boy with no complaint. Examination of the head, neck, and chest was essentially negative. The abdomen at this time was soft and not tender. No masses or nodes could be felt, nor was the liver or spleen palpable. The extremities were normal, and the neurological examination was negative. It was felt at this time that he might have had a subacute appendicitis. The urine examination was negative except for a trace of albumin and 1 to 3 white blood cells. The blood cell count showed R. B. C. 5,130,000; W. B. C. 5,650; polymorphonuclears 69%, lymphocytes 30%, basophiles 1%.

He remained asymptomatic for two days except for slight abdominal pain in the right lower quadrant, noticed particularly after eating. While he was being prepared for a gastrointestinal series, he suddenly had severe pain in the lower right abdomen, vomited greenish fluid, and collapsed. He was returned to his bed, pale and with cold and clammy skin. His pulse was good, and an enema gave effectual returns. Examination of his abdomen revealed at this time a deep localized tender mass in the right lower quadrant which seemed attached to and movable with the cecum. It was now felt that the patient had a localized appendiceal abscess adherent to the cecal wall, and a laparotomy was advised.

With the patient under gas-oxygen-ether anesthesia the abdominal cavity was opened.* An intussusception of small bowel was found entering through the ileocecal valve. The intussusception extended about 3 in. (7.6 cm.) beyond the ileocecal valve, forming a globular mass the size of a lemon. The intussusception was reduced, and after the reduction a mass appeared about 16 in. (40.6 cm.) beyond the ileocecal junction and in the small bowel. It was about half the size of a golf ball and extended into the lumen of the bowel. There were a large number of mesenteric glands that were edematous throughout the lower segment of the mesentery. They were discrete and appeared as if they were secondary to an inflammatory involvement of the small bowel. Between clamps, a V-shaped segment of the mesentery and about 6 in. (15.2 cm.) of the

* Dr. Benjamin Sherwin, attending surgeon, Lebanon Hospital, performed the operation.

Fig. 1.—Gross photograph of surgical specimen showing mushroom-shaped pedunculated lymphosarcoma projecting into the lumen of ileum.

small bowel about 2½ in. (6.4 cm.) on either side of the mass was resected. Lateral anastomosis was established. Several mesenteric nodes were also removed.

The pathologist's report † follows.

Gross Specimen.—Received a portion of small intestine, measuring 6 cm. by 4 cm. by 1 cm. The serosa is dark reddish-purple in color. It is smooth and glistening and does not show any external evidence of infiltration by tumor. The wall of the bowel appeared to be of normal thickness; however, there is a depressed area about 1 cm. in diameter on the serosa which, after the bowel is opened, is seen to correspond to a circumscribed mushroom-shaped papillary tumor, measuring 2 cm. by 2 cm. by 1 cm. in greatest dimensions. The center of the surface of this lesion is umbilicated, with radiating clefts which extend to the periphery and subdivide the mass into lobules. There is a short broad base of attachment to the wall of the bowel. The rugae in the area just proximal to the mass are flat. Five centimeters of uninvolved bowel are present on the proximal side and 1 cm. of uninvolved bowel on the distal side of the tumor. On section, the appearance is homogeneous, grayish-white, and cellular, and the lesion appears to extend into the wall of the bowel, almost reaching the serosal side.

† The pathological report was rendered by Dr. Joseph Ehrlich, Director of Pathology, Lebanon Hospital.

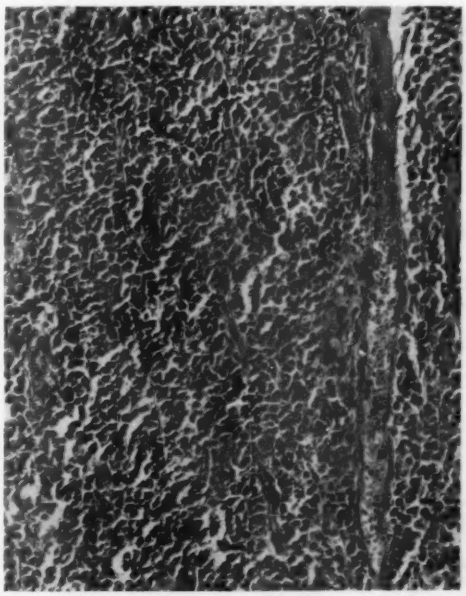
Microscopic Findings.—Section reveals this tumor to be composed of immature cells belonging to the reticulum cell and lymphoblast group. The cytology is characterized by the usual atypical features, including frequent mitotic figures. The lesion penetrates the muscularis of the intestine but does not reach the serosa. It ends abruptly at the proximal and distal edges, where there is a sharp transition to normal mucosa. In several fields frequent eosinophilic leucocytes are present. In areas on the surface, the mucosa is absent and is replaced by a thin layer of inflammatory exudate and necrotic cells. The neighboring mucosa contains several aggregates of normal lymphoid tissue.

Gross and microscopic diagnosis: Polypoid circumscribed lymphosarcoma of the ileum.

The postoperative course was uneventful, and the patient healed well. He was discharged on May 5, 1947, eleven days after his admission. The x-ray treatment was given by Dr. Maurice Lenz. The total dosage was 2,600 r at the tumor, from May 16, 1947, to July 21, 1947. All the treatment was given through one anterior central abdominal field, 20 cm. by 20 cm. The technical factors were 250 kv.; 50 cm. T.-S.D.; 0.5 mm. Cu filter; 31.6 r min.; H.V.L. 2 mm. Cu.

The boy continued to do well and received no further x-ray therapy. He has gained in height and weight in a normal fashion, and frequent periodic examinations to the present time have failed to

Fig. 2.—Photomicrograph of lymphosarcoma of the ileum. The tumor is composed of immature cells belonging to the reticulum cell and lymphoblast group; reduced ⅔ from mag. × 250.



reveal evidence of recurrence or metastasis elsewhere in his body. Clinically, one may assume that this boy has been cured, since it is now seven years since his operation.

Lymphosarcoma is a neoplasm which develops from lymphoid and reticuloendothelial cells. It commences in a focal area or group of nodes. Lymphosarcoma of the small intestine occurs most often in the ileum.[3] Involvement of the mesentery may produce a disturbance in the motor function. There may be diarrhea, occasional bleeding, dilatation of the affected intestinal loop, and, more rarely, narrowing of the lumen. Partial intestinal obstruction may be present, and loss of normal mucosal pattern may be visible on the roentgenogram. Intussusception may occur, especially if the lymphosarcoma is near the terminal ileum. In a report of five cases of lymphosarcoma of the bowel in childhood, Cutler[4] states that three of the five had symptoms and findings of intussusception, and in two cases involving the small intestine, the lesion was found in the terminal ileum.

Preoperative recognition of lymphosarcoma of the intestine in childhood offers many difficulties. In adults, bowel lesions of this type are usually confused with carcinoma until histologic examination clarifies the issue. The extraordinary rarity of carcinoma of the bowel in children is of considerable aid in the differential diagnosis. Probably the conclusion that a neoplasm of the intestinal tract in a child or infant is lymphosarcoma until proved otherwise is justifiable.

The impression is gained by some authors[5] that lymphosarcoma arising in the gastrointestinal tract often tends to remain localized to this tract and to the regional lymph nodes. While there is strong suggestion that some of these cases show recurrence after excision of the local tumor mass, they have not accumulated as yet sufficient evidence to support this contention.

Sufficient radiation can be delivered to lymphosarcoma localized in an organ or an area of involvement to halt growth, but if the disease is not localized, the chance of profoundly affecting it is not great.[6] Lymphoid tissue has a greater tendency to disseminate more easily than many other neoplasms and has a much greater sensitivity to radiation. No other tumor responds so rapidly to x-ray therapy as does lymphoid tissue.[7] The possibility of cure should therefore be greater for lymphosarcoma than for other neoplasms.

Radioactive therapy of localized lymphoid tumors must be directed to all palpable nodes and the surrounding tumor bed area.[8] In addition, it has been found that localized lymphoid tissue of the gastrointestinal tract is the most amenable to surgical treatment. On the other hand, treatment of the generalized disease is palliative in most cases. If the disease is adequately treated while localized, a significant number of patients will survive five years. Treatment of lymphosarcoma with radioactive phosphorus has been disappointing. Nitrogen mustards have not been satisfactory and should not be used for localized disease, since x-ray or surgical treatment is best here.

Since success depends on the eradication of the tumor while it is localized, Stout[9] feels that physicians should be instructed to believe in the possible curability of lymphosarcoma, in order that they may recognize it earlier and institute treatment during the period of curability.

SUMMARY

A case is presented of lymphosarcoma involving the terminal portion of the ileum in a 10-year-old boy. The lesion was widely resected, and this was followed by large doses of x-ray therapy. After seven years, the boy has grown normally and is in good health. We may assume that a cure has been affected in this otherwise highly malignant condition.

15 W. 81st St.

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RETROBULBAR NEURITIS

A Complication of Infectious Mononucleosis

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IN A REVIEW of the literature, there is a paucity of reports of optic nerve complications associated with infectious mononucleosis reported in children. Since 1931, when Johansen [1] and Epstein and Damashek [2] first reported central nervous system involvement in infectious mononucleosis, there have been few reports of this complication in children. Geliebter,[3] in 1946, was the first to describe central nervous system involvement in a child. This complication was encephalomyelitis. Recently, Karpinski [4] reported four cases of central nervous system complications of infectious mononucleosis occurring in children. Although cases of optic neuritis,* encephalomyelitis,† and serous meningitis [9] as complications of infectious mononucleosis have been reported, we have been unable to find any case of retrobulbar neuritis associated with infectious mononucleosis. Therefore, we feel it is of interest to report the first case of this complication.

REPORT OF A CASE

S. J., a white boy 6 years of age, developed an upper respiratory infection with a mild pharyngitis, malaise, and a low-grade fever, with a temperature of 100.6 F. (38.1 C.). He continued to run this low-grade fever for two weeks and then developed generalized lymphadenopathy, including the anterior and posterior cervical, axillary, and inguinal nodes. The spleen was palpable 3 cm. below the left costal margin. The rest of the physical findings were essentially normal except for mild injection of the mucous membranes of the nose and throat. Two weeks later, the spleen had receded to normal and the axillary and inguinal adenopathy had disappeared. However, he still had some residual cervical lymph node involvement. The low-grade fever continued until the heterophile agglutination became normal.

His past history revealed that he was subject to numerous bouts of recurrent bronchitis. He had scarlet fever at 4 years of age, three weeks after removal of his tonsils and adenoids. After this, he developed a pyelonephritis. After recovery from the latter, he ran a low-grade fever associated with poor appetite and loss of weight. This finally cleared after a few weeks. During this illness all studies were normal.

Laboratory studies, present illness: hemoglobin 85%, or 11.7 gm.: RBC 4,000,000; WBC 10,650; polymorphonuclears 33%, (segmented forms and nonsegmented 20%, 13%), lymphocytes 60%, monocytes 7%, with 8% atypical cells of the Downey type. Routine urinalyses and urine cultures were negative. Agglutinations for typhoid, paratyphoid, Brucella abortus and Proteus OX-19 were negative. The Paul-Bunnell test was positive in 1:224 dilution. This later rose to 1:896 and gradually decreased to normal.

Treatment consisted of penicillin and oxytetracycline (Terramycin) for two weeks, together with tripelennamine (Pyribenzamine) and supportive therapy.

While his heterophile agglutination reaction was returning to normal four weeks after the onset of his illness, he developed blurred vision. Neurological examination at this time showed that the other cranial nerves were not involved. There was no Brudzinski, Kernig, or Babinski sign present. Patellar and Achilles reflexes were normal. There was no nuchal rigidity.

A detailed ophthalmologic examination done at this time revealed the following findings: The vision in the right eye was 20/70 and the vision in the left eye was 20/20 —1. The pupil reaction was normal in both eyes. The corneae and conjunctivae were normal. Finger tension was normal, and the extraocular muscles were normal by

* References 5, 6, 9, and 16.

† References 7 and 8.

ductions, versions, cover-uncover, and alternate cover tests. The fundi of both eyes were normal. The optic discs were also normal. Retinoscopy using 1% homatropine was + 1.25 sph. ⊂ + 0.25 cyl. ax 90 (O.D.) and + 1.25 sph. ⊂ + 0.50 cyl. ax 90 (O.S.). Visual field examination by tangent screen was performed after the cycloplegic action subsided. The visual fields were normal when a 1 mm. white test object was used. However, there was a relative central scotoma of 3 to 4 degrees for a 3 mm. red test object in the right eye. There was no central scotoma for red in the left eye. Tests for blue and green were indefinite. Repeated tests gave similar findings. This condition gradually subsided over a period of months. A recent examination, one year later, revealed the following: The child's vision in the right eye was 20/20 —2 and in the left eye 20/20 —1, and there was no central scotoma. The fundi and visual fields were normal. The child accepted noncycloplegic correction of + 0.50 sph. ⊂ + 0.25 cyl. ax 90 in each eye, but glasses were not prescribed.

COMMENT

Although Emil Pfeiffer [10] in 1889 first described infectious mononucleosis and labeled it "glandular fever," he may have been preceded by Filatov,[11] in 1885, who described a similar syndrome, which he called "idiopathic lymphadenopathy." Downey and McKinlay,[12] in 1923, described the characteristic blood picture of "acute lymphadenosis," thus separating this entity from acute lymphatic leukemia. In 1932, Paul and Bunnell [13] demonstrated the heterophile antibodies in the blood of patients with infectious mononucleosis. It was not until 1931 that Epstein and Damashek [2] in this country and Johansen,[1] abroad, first noted central nervous system involvement in infectious mononucleosis. Since then many reports of this complication have been noted in this disease‡. Most of the above reports concerned adults. Silversides and Richardson [15] divided the neurological complications into four groups: (*a*) serous meningitis, (*b*) encephalitis and myelitis, (*c*) neuritis, and (*d*) polyneuritis. An acute polyneuritis similar to the Guillain-Barré syndrome has been described associated with infectious mononucleosis.§ Of course, there are many other causes of Guillain-Barré syndrome.[20]

According to Wintrobe,[21] headache and blurred vision are the commonest neurological complication. In 1950, Bernstein and Wolf,[6] in reviewing the literature, found 46 cases of infectious mononucleosis associated with central nervous system involvement. There was complete recovery in 85% of these cases. The involvement of the central nervous system in their series was less than 1%. Of 46 cases reported, 4 had blurring of vision, 1 had optic neuritis, 2 had papilledema, 1 had bilateral external rectus palsy with diplopia, 1 had paralysis of upward gaze and loss of convergence, 1 complained of diplopia, 1 had nystagmus and third-nerve paresis on the right, 1 had ptosis alone, 1 had slight ptosis of the right eyelid with blurred vision, and 1 had paresis of the left internal rectus with diplopia. Hubler and co-workers [22] described a case of bizarre encephalitis in which both eyes deviated to the right. Bonynge [5] reported a case of a 14-year-old girl with infectious mononucleosis who developed a real optic neuritis with hemorrhages and exudates. The latter case was improved with dimercaprol therapy, as recommended by Furmanski.[23] Bower || felt that gamma globulin was very efficacious.

Tanner,[26] in a recent article, divided the ocular manifestations of infectious mononucleosis into two groups: 1. Those possibly due to direct involvement of the eye and its adnexa by the characteristic pathologic lesion of infectious mononucleosis. In this group he includes conjunctivitis, eyelid and periorbital edema, episcleritis, uveitis, optic neuritis, papilledema, retinal edema, and retinal hemorrhages. 2. Those affecting vision and the neuro-ophthalmologic apparatus apparently due to a more remote lesion, most commonly involving the central nervous system. He includes extraocular muscle palsy, ptosis, nystagmus, disturbances of conjugate deviation, scotoma, and hippus.

Ashworth and Motto [27] reported a case of infectious mononucleosis complicated by

‡ References 6 and 14.

§ References 16 and 19.

|| References 24 and 25.

bilateral edema of the optic nerve and retina with no evidence of meningitis or encephalitis. Blaustein and Caccavo [28] described a case complicated by bilateral papilloretinal edema. Piel and co-workers [29] reported a case which involved the central nervous system with papilledema with a minimal amount of visual difficulty, but felt it represented a primary optic neuritis rather than evidence of intracranial pressure. With so many cases of optic nerve involvement being described in the literature, retrobulbar neuritis can certainly occur in the course of infectious mononucleosis.

The neurologic symptoms when they occur in infectious mononucleosis follow no definite pattern.[30] They may appear before the disease manifests itself. In other cases, the blood picture, lymphadenopathy, and neurologic manifestations may appear and subside simultaneously. In still other instances, the disease may subside and the neurologic symptoms appear later.

SUMMARY

The first reported case of retrobulbar neuritis as a complication of infectious mononucleosis is presented.

A brief summary of ocular manifestations occurring in this disease is given.

5365 Diamond St.

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Progress in Pediatrics

JUVENILE DELINQUENCY

An Over-All View

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THE NATURE OF THE PROBLEM

JUVENILE delinquency increased again in 1953 for the fifth consecutive year and is now at an all-time high. An estimate made from the report of juvenile courts throughout the country received by the Children's Bureau puts the number of children brought to the courts because of delinquency * in 1953 at about 435,000, exceeding the previous high of 400,000 cases dealt with in a single year during World War II.

The increase in delinquency cases for the five-year period 1948-1953 was 45%.

Increase in Delinquency.—Another aspect of the problem is that less densely populated areas of the country seem to be experiencing as sharp or even sharper increases than the national average. The courts serving jurisdictions of less than 100,000 persons showed an increase of 41% between 1948 and 1952, compared with a 29% increase for the nation as a whole. The increase for 1952-1953 was the same for all courts. It is clear from this that juvenile delinquency is not merely a "big-city problem." [1]

The increase in delinquency does not merely reflect an increase in the total child population. The increase, for example, of 45% in the number of children who came before juvenile courts between 1948 and 1953 compares with an increase of only 5% in the number of children in the country who were of juvenile court age (generally 10 to 17 years). In other words, the percentage increase in the number of delinquency cases was nine times as great as the percentage increase in the child population from 10 to 17 years of age between 1948 and 1953.

The prospect for the future is even more serious. The Bureau of the Census predicts that we shall have 50% more boys and girls in this 10- to 17-year age group in 1963 than we had in 1953. The babies born during and after World War II are growing up. Even if by extensive and strenuous efforts we are able to keep the rate of delinquency where it is today, we might well have an increase in the number of delinquent children of almost 50%, since, although the proportion of children who become delinquent would remain the same, the aggregate number of children would be so much greater.[2]

Commissioner of Health, City of New York (Dr. Baumgartner), and Director, Special Juvenile Delinquency Project, United States Children's Bureau (Mr. Beck).

Read in the symposium on Emotional Problems of Children as They Contribute to Juvenile Delinquency before the Joint Meeting of the Section on Pediatrics and the Section on Nervous and Mental Diseases at the 103rd Annual Meeting of the American Medical Association, San Francisco, June 25, 1954.

* The reliability of figures on juvenile delinquency is often questioned. The number of children who actually come before a juvenile court is generally considered the best estimate.

Not only is delinquency on the rise, but children are committing serious delinquencies. Federal Bureau of Investigation reports show that in 1953 juveniles under 18 committed 45% of all auto thefts, 49% of all burglaries, 16% of all rapes, 5% of assaults, and 4% of homicides.[3] One can speculate concerning the serious nature of the performance of today's delinquent. Is he just a more vicious character, or have technological developments made differences? Is the switch-blade knife of today yesterday's slingshot? Does the availability of the "hot-rod" lead to thefts unavailable to the lad who climbed the neighbor's apple tree for the joys of stolen fruit?

Or is the rise in juvenile delinquency merely part of a general trend? During 1953 there were more juvenile delinquency and more adult crime than ever before.

The majority of the boys and girls who come before the juvenile court for delinquent behavior are between 15 and 17 years of age. A recent study of 500 delinquent boys showed that nine-tenths of the children who came before the courts were having marked difficulty adjusting to normal social life before they were 11 years old; almost half of the group were showing noticeable signs of becoming delinquent at the age of 8 years or younger.[4]

There are 1,250,000 children who come to the attention of police officers each year because of some alleged delinquency. About one-fourth of that number are referred to the juvenile courts. Many youngsters, of course, commit delinquent acts which do not become a matter of official record. In a study of 114 boys from an underprivileged area, it was found that during a four-year period extending from their 11th to 15th birthdays, these boys had committed a total of 4,400 minor offenses, such as truancy, petty stealing, trespassing, running away, and sneaking into movies. Only 27, or 0.6%, of such minor offenses became a matter of court complaint. Of 616 serious offenses, such as breaking and entering, larceny, assault, drunkenness, and sex offenses, only 68, or 11%, became a matter of court complaint.[5]

Failure to Prevent.—The foregoing facts seem to demonstrate that the combined efforts of parents and professionals and our society have not been sufficient to hold back a rising rate of delinquency.

A number of studies have tried to ascertain the effectiveness of certain provisions—child guidance clinics, recreation, friendly counselling, and housing—as preventives of delinquency. Because of the difficulties inherent in social science research none of these studies is above criticism reflecting upon the accuracy of its findings. Some are extremely vulnerable to such criticism. Taken as a whole, however, these studies would appear to indicate that no single measure considered in and of itself can be taken as an effective preventive device.

In considering the limitations of our efforts to prevent delinquency, it must be noted that while our total investment of time, energy, devotion, and money in services to children is very high, there is no single community in which all of the components of a total program for children meets accepted standards. Even in those communities where there is an abundance of services, such services may not be organized in the manner most likely to produce results.

Failure to Treat.—The wealth of data indicates that if our total provision for prevention of delinquency can be termed inadequate, then a much stronger adjective must be used to describe our provisions for treating the delinquent child. Our agencies for helping delinquent children are doubly inadequate; there are not enough of them, and those we have do not offer the right kind of service.

In nearly half of our communities with populations over 20,000, not even one officer is assigned to work with children. Yet a recently completed study of police

services for children demonstrates that the presence of such officers makes it possible to reach children before they commit serious delinquencies and keep them out of jail.[6]

A majority of the counties in the United States hold certain children in jail while awaiting final court action. One hundred thousand children are thereby exposed to an atmosphere conducive to delinquency each year.† This, despite the fact that two such youngsters have been murdered by their contemporaries in such jails in the past decade. Such children should be held in detention homes especially designed to meet their needs. Only 200 such homes exist in America.‡ Two thousand five hundred juvenile courts have need of them.

Judges in these 2,500 courts have broad powers to act in the interest of children. Yet many have no training in law or any other professional discipline. Probation officers trained in social work are required to assemble social and psychological information to aid the judge in making dispositions. But one-half of the juvenile courts have no probation service. Those which do have service receive it from probation workers who are substantially untrained. Four out of 10 have no college degree. Only 1 out of 10 has completed social work training.[7]

Of more than 180 public institutions in America, one-third have populations in excess of 200 children. Considerably less than a half have the services of a psychiatrist, full or part-time. One-third do not have a social worker on staff—trained or untrained.§

In most cases, communities are maintaining agencies that do no more than restrain and lock up young people. These agencies make no attempt to find out the causes of these children's misbehavior and help them correct it. Yet therein lies our only hope of preventing these young delinquents from going on to become the criminals of tomorrow. Certain studies have shown that more than 5 out of 10 young people who appear before our juvenile court today go on to commit one or more serious crimes for which they will be convicted as adults. A large majority of the inmates of our prisons for adults were once juvenile delinquents.

Crime costs America $20,000,000,000 per year.‖ About $200,000,000 is spent on police, courts, detention, and institutionalization of juvenile delinquents.¶ Yet much of this money is wasted—too little, too late. This is false economy. Funds invested in a nation-wide effort to curtail delinquency today would, even if that effort were only moderately successful, save us many times the amount invested in future deduction of our tremendous crime bill—as well as in young lives turned from twisted and antisocial behavior to responsible citizenship.

UNDERSTANDING THE PROBLEM

Any attempt to cope with juvenile delinquency on a national basis must, of course, be based on knowledge now available as to the causes of the problem. Much research has examined the traits of the delinquent child rather than the cause of delinquency. Such enumerations have misled many persons to mistake the traits found to be

† Children's Bureau estimate.

‡ The estimate of the number of detention homes in the United States is based on data used by the Census Bureau in connection with its 1950 decennial census.

§ Unpublished Children's Bureau survey.

‖ Testimony of J. Edgar Hoover, Chief of Federal Bureau of Investigation, before Committee on Appropriations, House of Representatives, Eighty-Third Congress, second session (1954).

¶ Children's Bureau estimate.

common among groups of delinquent children to be the causes of their delinquency. This, in spite of the fact that research workers have dispelled the notion that delinquency is caused by any one factor, such as poverty, broken homes, membership in a minority group, or membership in a gang. No one, of course, denies that any one of the factors mentioned can play a predominant role as a contributing or precipitating factor to the delinquency of particular groups of children. That there is a continuous need for basic research into cause and means of prevention is all too apparent.

The Culture.—While our knowledge of cause is certainly not precise, there is some knowledge which gives sound direction to preventative and treatment efforts. Early in the century came the first of a series of studies clearly demonstrating the association between delinquent behavior and underprivileged neighborhoods.[8] Deeper significance was given to this discovery by the research of cultural anthropologists who determined that the standards, ideals, goals, and ways of behavior differed significantly between people who lived on the "right" side of the tracks and people who lived on the "wrong" side of the tracks. Youngsters who lived in slum neighborhoods did not receive from their parents and from the cultural milieu in which they lived the same kind of impetus to repress open expression of hostile behavior that children received in neighborhoods which were economically and socially favored.[9]

Such an analysis of the problem, of course, did not shed a great deal of light on the question of why a particular child living in an underprivileged area became delinquent while the neighbor's child did not. Early studies of this phenomenon, comparing delinquent and nondelinquent children of the same family, suggested that there was a difference in the emotional needs of the children.[10] More recent and more precise studies comparing delinquent and nondelinquent children in underprivileged areas determined that the delinquent children, to a far greater extent than the nondelinquent children, are reared in homes of little understanding or stability.[4]

The Individual.—Delinquent children in slum neighborhoods, to a far greater extent than nondelinquent children in slum neighborhoods, are reared by parents who are usually unable to be effective guides and protectors. Such parents are not desirable sources for emulation, and because of their own limitations, they cannot lay the foundations for a consistent, well-balanced, and socially normal personality during the early stages of the character development of the child.

In other words, the seeds of what, for lack of a better term, can be called "character malformation" are sown during the first few years of life. When these seeds are sown in a family living in an underprivileged area, it is highly probable that the youngster will become delinquent because of the lack of repressive forces in the neighborhood and the inducement to delinquency. It is possible that when these seeds are sown in a family living in a more favored community, the repressive forces on expression of antisocial behavior will be great enough to keep the child from becoming delinquent but will result in the development of neurotic symptoms, taking a great toll on the person but a less obvious and direct toll on society.

The Society.—In recent years the problems of juvenile delinquency has assumed a new dimension not yet adequately explored by social science research. The existence of this dimension is attested to mainly by the testimony of persons all over the country who are actually working in positions where they have occasion to observe the conduct of young people. Such observers report that in America today there is a wave of delinquency often manifested by excessive vandalism, which cuts across class and caste and seems to have little reference to the individual background of

the child. The existence of such a wave of delinquent behavior is also substantiated by reports of referrals to juvenile courts which show the sharp rise of delinquency in the less densely populated areas where heretofore delinquency has not been a major problem.

It is possible to use research knowledge now available to illuminate the roots of the current situation. Since the incidence of character malformation in these times is high, and since children with character malformation living in communities with a high aggressive component are liable to turn to delinquency, then is it not possible that the wave of delinquent behavior today comes about because all children are living in a world where the aggressive component is high and tends to be unrestrained? This hypothesis is supported by the graph of the rate of juvenile court referrals which show positive correlation between the increase of delinquency and the time of war or international tension.

Support for this contention is found in the words of a 17-year-old high school boy who, in writing on the subject of juvenile delinquency, had this to say:

> The apathy of the American public toward graft and corruption which runs so rampant is passed on to my generation, which notes the success of illegality, and sees approbation for this method of easy money; the resentment which we feel when we are castigated for doing the very things our elders do, or for wasting our time when there is comparatively very little to do that does not cost an excessive amount of money; and, most of all, the feeling of complete worthlessness which comes over us when we realize that our life span may be determined by the overall political picture as seen by one or the other of the two major parties—that the value of our life is counted in so many votes—all these are responsible for the ever more prevalent devil-may-care attitude which results in the increase of delinquency. . . .
>
> I believe you should wage a campaign—or perhaps a better word would be crusade—to eradicate the factors leading to delinquency, to instill within my generation a sense of community responsibility, coupled with the feeling that we are wanted by the community. We would like to be able to feel that we are not tolerated as pesky nuisances—abided by only because in a few years we can be sent off to war—but rather that as a group we are wanted and can be of use, not only to the nation by bearing arms, but to our States and home towns or cities.

This analysis of the current increase of delinquency has great significance for any plan to cope with the problem of delinquency. It reemphasizes the fact that delinquency is above all a social problem. It puts delinquent behavior in its proper perspective as a symptomatic response of youngsters to the world in which they live. It relates the hostile antisocial behavior of youth to the insecurity and hostility of adults. It demonstrates that although the trained technician has a vital role to play in particular programs for delinquent children, the problem cannot be solved simply by adding to our supply of experts and making them available to children with problems. It becomes apparent that to cope with this problem, a program must have an effect on the social and moral tone of communities in which children live. It is only in this atmosphere that the precise programs needed can produce results.

COMPONENTS OF A SUCCESSFUL PROGRAM

To achieve a measure of success, therefore, a program to curb delinquency must apparently have within it the potential of a social crusade which will change the attitudes of those involved about themselves, their neighbors, and their communities and thereby change the face of the community in which those persons live. Within this broad framework, one can designate four activities which apparently can be helpful in curbing delinquency. Briefly they are as follows:

(1) An expanded research program.

(2) Basic prevention through concern with the development of health personalities in all children.

(3) Early remedial work with those who become delinquent.

(4) More effective and less damaging treatment of the child after he has become officially delinquent.

Physicians, as citizens, working "solo" fashion with their own patients and working with other professionals in a team, have much to offer and should be better informed about all of these activities. Time allows for only brief comment concerning them.

Basic Prevention.—With some precision it has now been demonstrated that loving maternal care in a family home during the earliest years of childhood lays the foundation for healthy emotional development in adult life. Efforts of basic prevention must be concerned with those social programs designed to bolster family living, such as those which advance economic well-being and provide decent housing. Since the aim is to keep both parents together with a sense of family well-being, all measures designed to cope with the problem of chronic illness and physical disability, as well as those aimed in a more positive sense at health maintenance, are of particular importance. Individualized social and psychological services for families which may be threatened with dissolution warrant strengthening. Discussion groups for expectant parents in which mutual problems may be explored and the body of knowledge about child development drawn upon are useful. The doctor, in his individual contacts with patients, must become more skillful in helping parents foster sound emotional development in their children.

As the child grows, provisions which meet the changing needs of childhood must be available. Here again the bolstering of family life and the encouragement of family activities is mandatory. The church and its auxiliary activities, recreational services, and youth organizations have valuable contributions to make. Essential to success is the all-around betterment of the public school system. A good teacher in a small school with a sound program may well be the heart of the preventative effort.

Early Remedial Work.—Despite the most strenuous effort, certain children will develop in such a way that unless they receive individual attention they will become delinquent. It is not easy to determine which children showing early signs of maladjustment will become delinquent and which will develop some other symptom of deviation, so that so-called early remedial work will be of substantial benefit to all children, although instituted in the interest of curtailing delinquency.

A variety of prognostic devices, including that of simple observation, are available to determine which of a group of children is experiencing difficulty in making a healthy adjustment. As part of the program for remedial action, education in early problem detection must be provided for teachers, recreation workers, parents, and the like. Locating such children when no remedial help is available is, of course, stupid. Every community should make adequate provision for mental health services, group work services, and case work services to meet the needs of those who warrant attention. Particular emphasis must be given to the adaptation of such services so that they can serve those with a limited understanding of their problems and a limited desire to seek help.

Treatment.—The next phase of the delinquency control program is concerned with the child after he has become officially delinquent. Since the largest number of these children are handled by police officers without reference to any other agency, special attention must be given to the development of training programs for such officers. The establishment of juvenile units in police departments serving the more densely populated areas must be encouraged. The aim would be to promote con-

structive handling of the youthful delinquent by the police officer so as not to ingrain feelings of hostility against those in authority. If the officer is not bringing the child before the juvenile court and not simply releasing the child to the child's parents, he must have some facility to help in ascertaining the needs of the child and making a proper referral to the appropriate health or welfare agency.

Particular emphasis must be given to the eradication of the use of county jails and local police lockups to hold children while awaiting official action by the juvenile court. In their place must be substituted facilities so staffed and organized that detention in secure custody becomes the first step in treatment and in the acceptance of legitimate authority, rather than the first step on the road to criminality.

The juvenile court in many states requires restructuring so as to bring to its bench judges sympathetic to the needs of children, interested in their work, and capable of using the sciences of human behavior when they reach the point of determining what type of treatment is in the interest of the child. Such courts require the staff of well-trained probation officers which will enable the judge to leave many children in their own homes with expert guidance and supervision, rather than committing them to state institutions or placing them in foster homes. Attention must be given to the institutions for delinquent children which all too often are merely dumping grounds for children nobody wants and for whom no provision has been made. If these institutions are to do their job, they must have programs geared to the needs of children, and they must receive only those children who can benefit from their programs.

Coordination.—These activities are obviously interrelated. Ignoring this fact results in a great waste of service and seriously hampers effectiveness. A recent study in a metropolitan area having a wide variety of health and welfare services revealed that 40% of those services were concerned with only 6% of the families living in that area.[11] This and other studies have demonstrated that the problem of delinquency usually does not exist alone in a family, but that it is one of a constellation of maladjustments. Although such families may receive a lion's share of total service time available, they often do not receive substantial help. The variety and severity of their problems sometimes result in their being shunted from agency to agency. Frequently, no one agency has the resources and techniques for sticking by them and working intensely toward their readjustment in the community. Without proper coordination of services, not only may families be shunted from one agency to another, but the needs of certain families may be overlooked. Services are often spread too thin, or there are gaps between agencies. Sometimes there are weaknesses at an early stage of service that increase the burden at a later stage.

Providing effective and economical services and facilities requires some device for coordination, planning, and social action. Such a device may be found in the type of coordinating body which brings together the experts offering service and those people who must either through tax funds or voluntary contributions support the services. Without this kind of teamwork, it is not possible to get the support necessary. With this kind of teamwork, however, not only is it possible to improve particular programs, but an opportunity is offered whereby the maximum number of persons living in any given community can work with one another toward mutual goals. Thereby they become involved in activities which accent community interest rather than immediate self-interests. Thus, the coordinating device can be used to improve the moral fiber of the community—the social atmosphere in which children are raised.

This is the basic ingredient for the successful program to combat delinquency, for it is only in such a milieu that the professional experts can function effectively. Paradoxically enough, however, in creating such a milieu we decrease the need for such experts, since we create the climate in which delinquency and kindred social evils do not flourish.

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Scientific Exhibits

NEWER CONCEPTS OF INFANT RESUSCITATION AND POSITIVE-PRESSURE THERAPY IN PEDIATRICS

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MUCH HAS been written about the pros and cons of positive pressure in infant resuscitation. The present paper presents a new approach to the problem through the use of accurately controlled intermittent positive pressure. Simplicity and availability are emphasized in the development of an infant resuscitator which can safely achieve expansion of the atelectatic lung of the newborn, while supplying adequate oxygenation. It is but a step further to combine intermittent positive pressure with aerosol therapy in the treatment of other neonatal respiratory complications and in the treatment of asthma and chronic pulmonary conditions in older children.

I. Infant Resuscitation

A. The Problem of Infant Resuscitation

1. Infant Mortality

Modern medicine in increasing the longevity of mankind has decreased the infant mortality rate by approximately two-thirds during the 33-year period from 1915 to 1948 (32.0 deaths per 1,000 live births in 1950). The largest decline occurred in the second half of the first year (85%); deaths occurring during the first 24 hours declined less than one-third (roughly 35% of all deaths during the first year occurring in the first 24-hour period [Fig. 1]).[1]

From the Departments of Pediatrics and Engineering (Mr. Bennett, Consulting Engineer), Lovelace Foundation for Medical Education and Research.

Shown as a scientific exhibit of the Section on Pediatrics at the 103rd Annual Meeting of the American Medical Association, San Francisco, June 21-25, 1954.

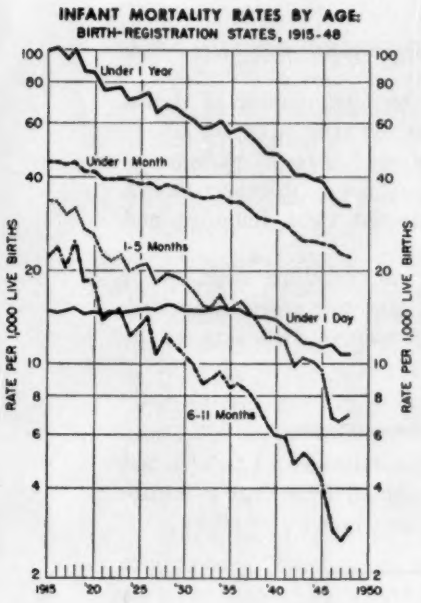


Fig. 1.—Trend in infant mortality (reproduced with permission from *Special Reports*, Vol. 35, No. 13, National Office of Vital Statistics, U. S. Public Health Service, Aug. 23, 1950.

Leading Causes of Death in Neonatal Period
(Per 1000 live births)

	Premie	FT	Total
Anoxia	0.4	0.2	0.6
Trauma	0.3	0.4	0.7
Malformations	1.0	1.0	2.0
Erythroblastosis Fetalis	0.2	1.0	1.2
Miscellaneous	0.4	0.3	0.7
No abnormalities	0.3	---	0.3
Pulmonary Pathology			
Hyaline Membrane	2.6	0.6	3.2
Other	1.1	0.2	1.3

Fig. 2.—Leading causes of death in the neonatal period (Potter [3]).

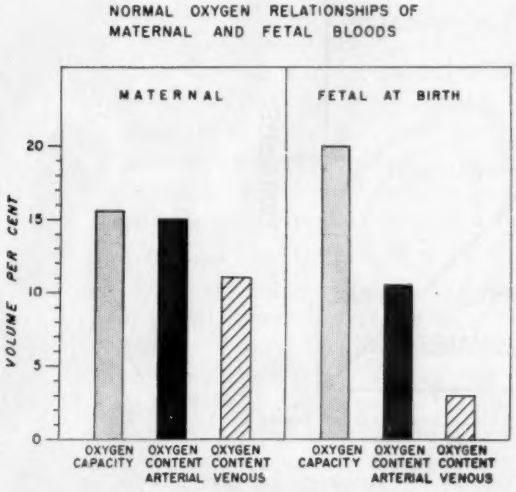


Fig. 3.—Normal oxygen relationships of maternal and fetal bloods (reproduced with permission from the *American Journal of Obstetrics and Gynecology).*

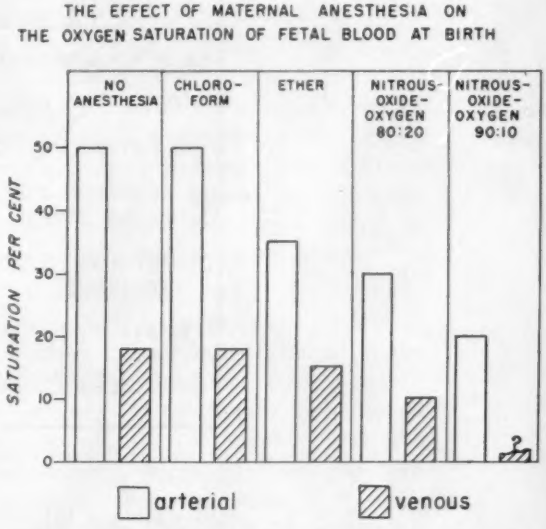


Fig. 4.—Effect of maternal anesthesia on the oxygen saturation of fetal blood at birth (reproduced with permission from the *American Journal of Obstetrics and Gynecology).*

ANOXIA

TYPE	CAUSE
A. Fetal	Maternal pulmonary or circulatory disease Over solicitous use of pre-partum medication Anesthetic gas replacing maternal oxygen Interference with placental exchange; obstructed cord.
B. Neonatal	Poor aeration of blood in lungs * from numerous central nervous system and pulmonary causes Right to left shunts caused by cardiac or circulatory malformations

*This anoxic anoxia (low tension throughout the capillary bed) is the type which is improved by the administration of oxygen.

Fig. 5.—Anoxic anoxia in the newborn (modified from Smith, C. A.: Am. J. Dis. Child. **79**:1-9 [Jan.] 1950).

Approximately 150,000 deaths, or one-tenth of the total number of deaths occurring in the United States annually from all causes and at all ages, are infants who die in association with the birth process. As Eastman [2] states so realistically, these are infants whose life expectancy is 62 years; 62 years, moreover, which span the prime of life, which span the decades of greatest vigor, initiative, and achievement.

When today's leading causes of death in the neonatal period are examined, it becomes evident that anoxia and pulmonary pathology constitute greater than 50% of the causes of death in this early period (Fig. 2). These two conditions are the most responsive to resuscitative measures.

2. Categories of Resuscitation

Babies requiring resuscitation fall into three categories:

a. Category I. The baby who exerts no respiratory effort at birth and requires expansion of unexpanded lungs and thoracic cage to initiate respiration or is unable to expand his lungs by his own effort

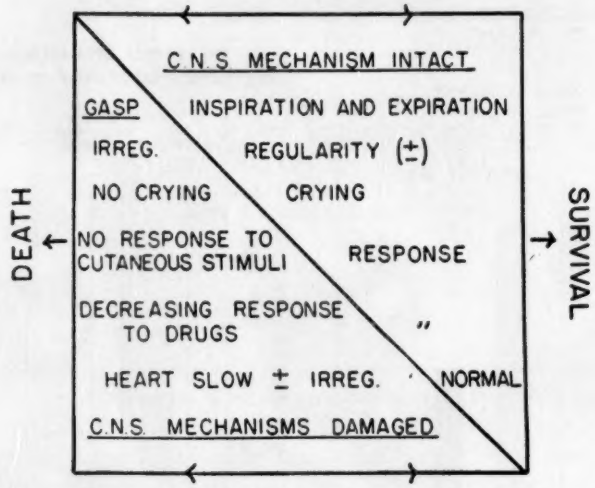


Fig. 6.—Respiratory onset at birth (reproduced with permission from Smith, C. A.: Am. J. Dis. Child. **79**:1-9 [Jan.] 1950).

b. Category II. The baby who breathes spontaneously but does not do well and usually has partial atelectasis

c. Category III. The anoxic baby who benefits by the administration of oxygen

3. Anoxia

There are many causes of fetal anoxia. Some of these are beyond the control of the obstetrician—premature separation of the placenta, prolapsed cord, uterine inertia, prepartum bleeding, etc. The obstetrician, however, can prevent oversedation with antepartum analgesics, which may lower an oxygen arterial content already only 50% of the fetal oxygen capacity (Fig. 3).[4] Together with the anesthesiologist, he can avoid the improper selection or use of anesthetic agents at delivery which depress this oxygen saturation even further (Fig. 4).

In spite of all precautions, anoxia is frequently encountered at delivery, and it is this fetal anoxic anoxia (Fig. 5) which most readily responds to resuscitative measures.

Respiratory onset at delivery follows one of two patterns: (1) CNS mechanism intact—respiration begins at birth as a rhythmic integration of inspiration followed by expiration frequently punctured by crying. These infants are strikingly responsive to cutaneous and proprioceptive sensations and need very little in the way of resuscitative measures. (2) CNS mechanism damaged—e. g., impairment by anesthesia, anoxia, trauma—respiratory onset is delayed; series of irregular gasps without a true expiratory component, with no response to cutaneous stimuli, and with decreasing response to drugs. These are the infants who need to have respiration established. The original inflation of any alveolus requires more force from the infant or from the physician in his behalf than do the breaths which follow.[6] Arrows at top and bottom of diagram (Fig. 6) indicate that the situation of any infant may move in either direction in the immediate postnatal period.

4. Abnormal Pulmonary Ventilation

a. Historical Background

The greatest challenge today in infant resuscitation is expansion of the unexpanded lungs in the infant who exerts no respiratory effort at birth or is unable by his own effort to expand his lungs. Aid to the infant who has breathed spontaneously but has partial atelectasis and respiratory distress is also a difficult problem. Wilson and Farber [6] demonstrated that positive air pressures greater than 25 cm. of water are necessary to expand the lungs of newborn infants. Smith and Chisholm [7] reiterated that pressures of 20 to 30 cm. of water are required to overcome the factors resisting the expansion of the human lung at birth and that the newborn infant, in struggling for a breath, can produce inflating pressures in excess of 40 cm. of water. Although the pressure necessary to expand the lungs and that which the newborn infant himself exerts may be pressure conditions of different natures, it still appears justifiable to approach the levels which the infant himself creates.

b. Development of the "Day Impulse Principle"

Barach and co-workers,[8] studying the cough mechanisms in adults, had shown that an increase in duration of pressure resulted in progressive inflation of the lungs. Working on this premise, Dr. Richard Day postulated that (1) injury to a lung arises from distention, not from pressure; (2) distention is proportional to pressure as well as to time the pressure is exerted, and (3) therefore, pressures applied for short intervals are probably safe to use in expanding the unexpanded newborn's lungs. He devised a simple laboratory apparatus, called the "impulsuflator," which could deliver positive pressures from 1 to 60 cm. of water over controlled time periods. In work with small young animals—namely, rats—lungs were artificially collapsed and studies carried out to determine the minimal and maximal positive pressures required to expand those atelectatic rat lungs.

Pressures up to 27 cm., applied over time periods as great as 1.5 seconds, such as used in current methods of resuscitation, did not expand these atelectatic rat lungs. Expansion was achieved with pressures of 30 cm. or over at this time interval. Higher pressures were required to achieve similar expansion when shorter time intervals, simulating an infant's initial inspiratory gasp were used, i. e., 40 cm. required when time interval was 0.15 seconds. The conclusions of Dr. Day and co-workers,[9] were that (1) rupture was probably the result of overdistention, whether this distention be produced by too high a pressure or too long an application of a lower pressure; (2) a certain minimum pressure must be exerted to correct atelectasis; to employ this pressure safely, its duration must be limited, and (3) pressures exerted by normal infants are similar to the pressures

which have produced the best results in experiments on animal lungs. From this work was born the "Day impulse principle," or the use of high pressures over short time periods. Subsequently, Wilson [10] has added that damage to the newborn lung is dependent not only upon the degree of pressure and the time it is applied, but also on the proportion of total lung volume that is expansible.

B. The Present Investigation of the Problem

1. Objectives in the Development of an Infant Resuscitator

The present investigation began in September, 1952, with the opening of the Pediatric Research Laboratory of the Lovelace Foundation for Medical Education and Research, in Albuquerque, N. Mex. At the start of these studies at the Foundation, the following objectives were outlined as requisites in the development of an infant resuscitator:

a. Expansion of nonexpanded (atelectatic) lungs of newborns who do not initiate their own respirations or expansion

b. Assistance in correction of partial atelectasis in newborns with respiratory distress

c. Oxygenation for anoxic newborns, with provision for variation in gaseous mixtures

d. Provision for an attachment allowing deliverance of moisture, wetting agents, and aerosols

e. Promotion of drainage of upper and lower respiratory tracts

f. Simplicity and availability of infant resuscitator

2. Laboratory Studies with Intermittent Positive Pressure

Teamwork was considered imperative in carrying out such a program and in the successful development of an infant resuscitator which was to meet the objectives outlined. The Foundation's team consists of obstetricians, anesthesiologists, pediatricians, surgeons, roentgenologists, pathologists, respiratory physiologists, engineers, and medical illustrators. Each is an integral part in answering the problems which have arisen in the present investigation.

IPP (intermittent positive pressure) was believed to be the answer to the first two objectives. Laboratory studies with balloons and animals were repeated. Realizing the importance of determining the response of human infant lungs to positive pressure forces, atelectatic lungs after neonatal deaths were removed within two to four hours of death, and postmortem studies were carried out to determine the elasticity of the human lung and the forces required for expansion.

For postmortem study of the effects of IPP, three methods were used. Pressure was applied to (1) lungs in closed chest, (2) lungs in situ but with chest wall open, and (3) lungs removed from chest and suspended freely in air. In the first two methods pressure was applied via facepiece and catheter inserted into the trachea. Figure 7 shows *A* uniform expansion with 50 cm. IPP applied via facepiece to lungs of 4 lb. (1,815 gm.) premature infant (lungs in situ), *B* unexpanded lungs of 775 gm. premature infant, Twin A, as contrasted to C expansion of same lungs with 80 cm. IPP via catheter with lungs in situ in open chest, and D aeration of lungs of 665 gm. Twin B with application of 80 cm. via catheter to lungs in closed chest (pneumothorax occurred at 95 cm. pressure).

To study expansion achieved with varying pressures applied to the lungs of a 3 lb. (1,360 gm.) premature infant, the lungs were removed from the chest wall and suspended freely in air. The right main bronchus was tied off, and pressure

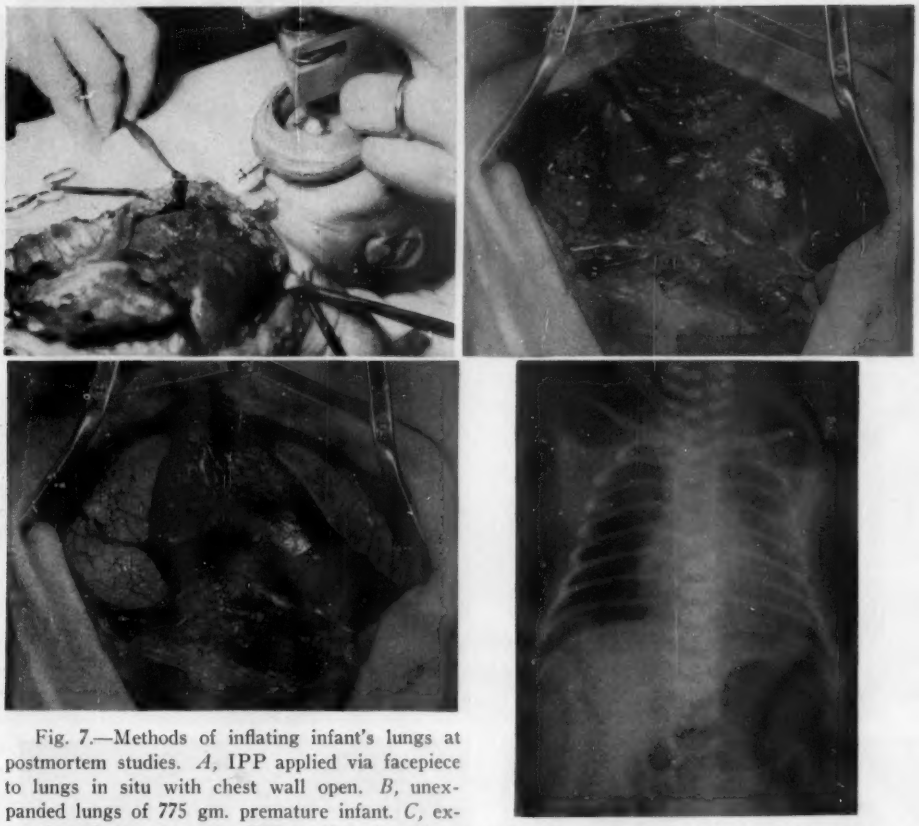


Fig. 7.—Methods of inflating infant's lungs at postmortem studies. *A*, IPP applied via facepiece to lungs in situ with chest wall open. *B*, unexpanded lungs of 775 gm. premature infant. *C*, expansion of same lungs by IPP applied via catheter in trachea. *D*, IPP applied via catheter in trachea to lungs in closed chest.

was applied via a catheter tied into the left main bronchus. This allowed contrast between the unexpanded lung and the lung receiving positive pressure. Simultaneous studies with a fine radiopaque aerosol delivered under pressure via the hand resuscitator outlined that portion of the lung which expands at certain pressures. Figure 8 shows *A* no gross expansion of left lung when 20 cm. positive pressure applied over 0.2 second interval, *AA* only the largest portions of the bronchial system outlined at 20 cm.; *B* beginning patchy aeration with 30 cm. of positive pressure over 0.2 second interval, *BB* some of alveolar system outlined by radiopaque aerosol now, as well as bronchobronchiolar system; *C* more uniform expansion achieved with 40 cm. positive pressure, *CC* more uniform outlining of entire respiratory system; *D* 50 cm. positive pressure required for adequate uniform expansion of lungs removed from thoracic cage, and *DD* entire lower respiratory system uniformly well outlined with 50 cm. positive pressure.

In a similar postmortem study in a 1,160 gm. premature infant, the left main bronchus was tied off and a catheter tied into the right main bronchus. A pressure of 40 cm. was applied over 0.2 second interval. Figure 9 shows microscopically *A* the compactness of the unexpanded left lung (low power); *B* the collapsed compact alveoli of the unexpanded left lung (high power); hyaline membrane is present

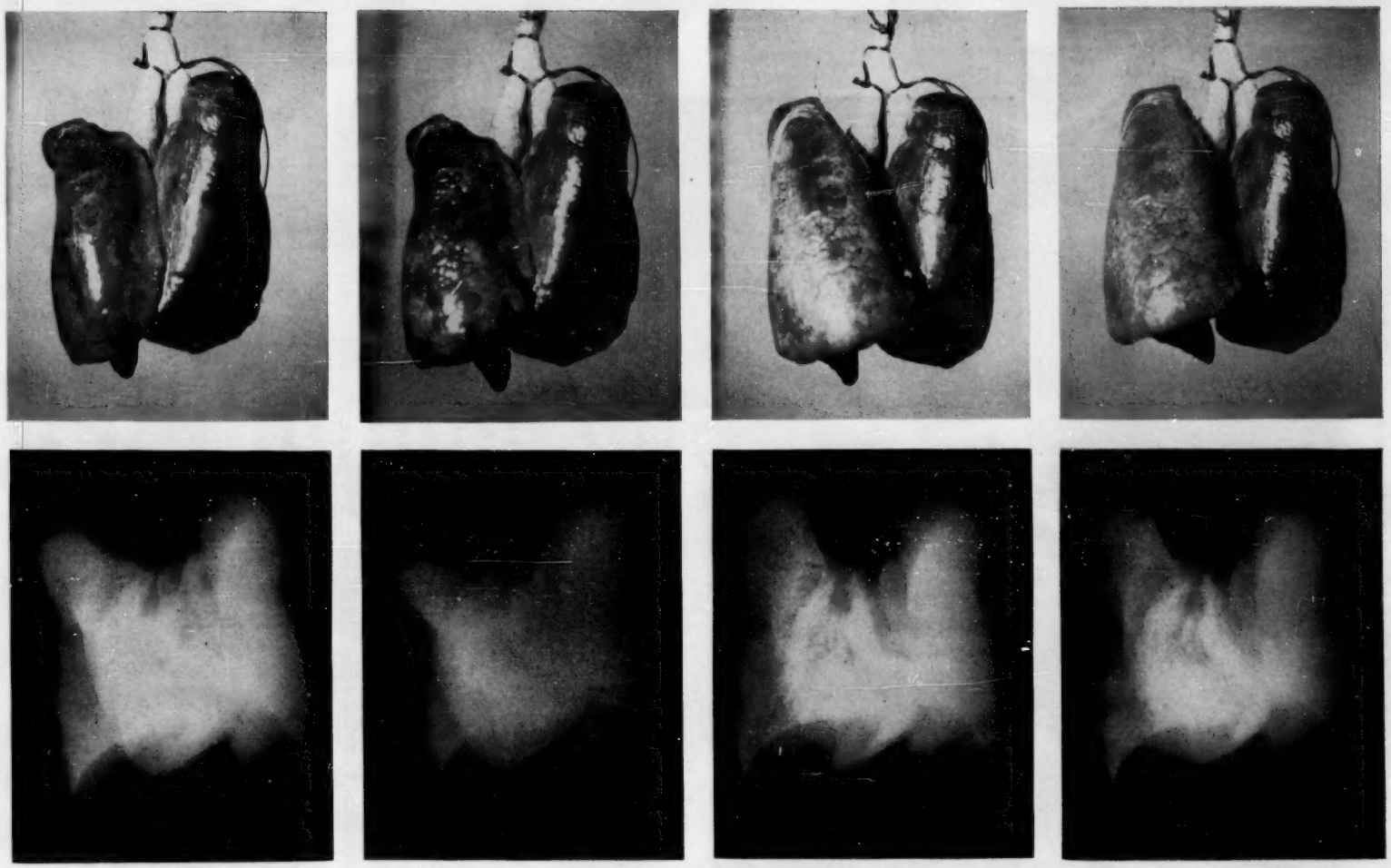


Fig. 8.—Gross pathological and x-ray studies of expansion of lungs of 3 lb. (1,360 gm.) premature infant. *A*, no gross expansion with 20 cm. positive pressure (lungs suspended in air). *AA*, radiopaque aerosol outlines largest portions of bronchial system at 20 cm. *B*, beginning patchy aeration with 30 cm. *BB*, alveolar and bronchobronchiolar system outlined with 30 cm. *C*, uniform expansion with 40 cm. *CC*, more uniform outlining of respiratory system with 40 cm. *D*, adequate uniform expansion with 50 cm. *DD*, entire lower respiratory system well outlined with 50 cm.

Illustrations are arranged as follows: *A*, *B*, *C*, *D*, top row, left to right; *AA*, *BB*, *CC*, *DD*, bottom row, left to right.

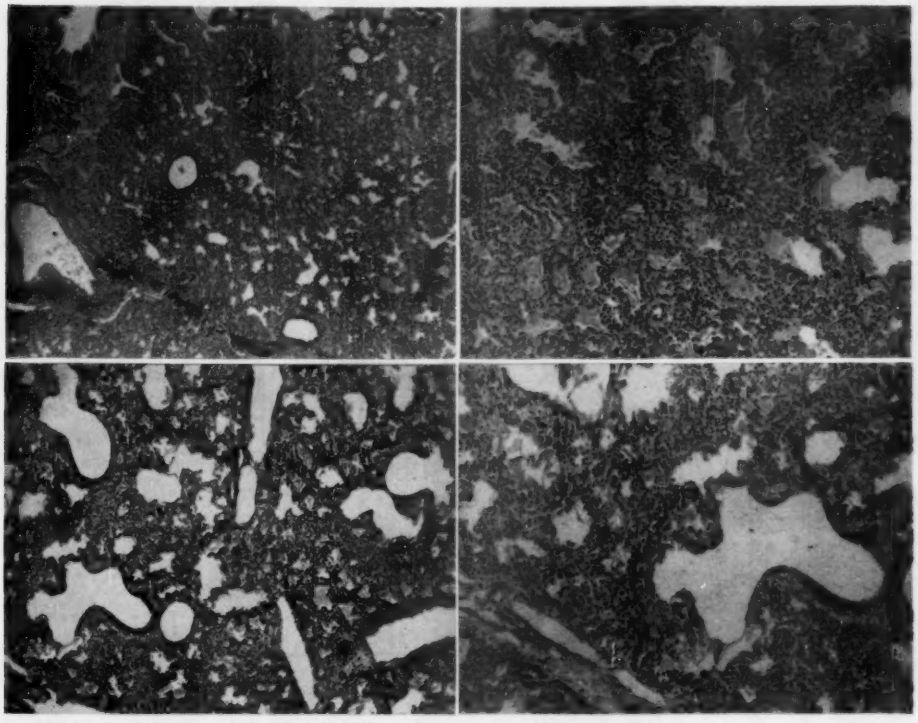
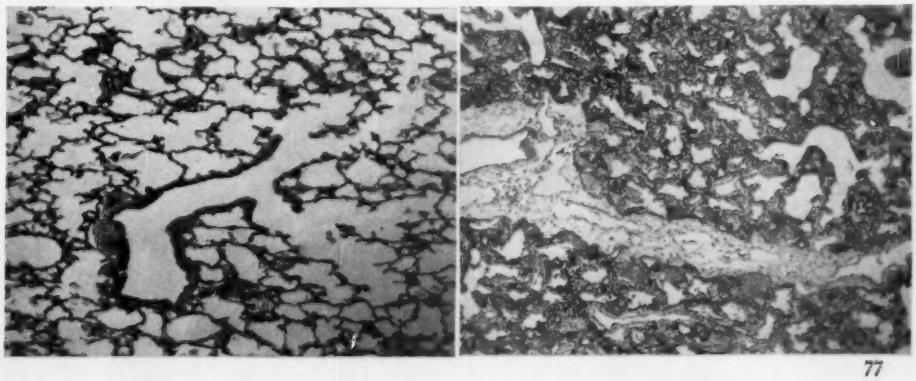


Fig. 9.—Microscopic study of expansion achieved in 1,160 gm. premature infant with 40 cm. pressure. *A*, unexpanded left lung (low power). *B*, hyaline membrane obstructing alveoli of unexpanded left lung (high power). *C*, some expansion of right lung with 40 cm. positive pressure. *D*, disruption or modificatior of hyaline membrane lining terminal bronchiole.

in many alveoli, not only lining the alveolar wall, but filling the alveoli and offering actual mechanical obstruction; *C* the right lung, to which 40 cm. pressure was applied showing some expansion—25 to 40%?—(low power), and *D* a terminal bronchiole distended by 40 cm. positive pressure applied to the right lung; some disruption or modification of the hyaline membrane lining this bronchiole has occurred, creating space for repiratory exchange.

Figure 10 shows *A* uniform complete expansion without evidence of injury at 60 cm. in a 5 lb. (2,270 gm.) premature infant dying at 18 hours from extensive

Fig. 10.—Microscopic expansion achieved with 60 and 80 cm. positive pressure. *A*, complete expansion without injury with 60 cm. *B*, interstitial emphysema with 80 cm.



intracranial damage and *B* microscopic damage in the nature of interstitial emphysema when 80 cm. positive pressure was applied to the lungs of the 775 gm. premature twin of Figure 7*C* (thin barium solution used in this study can be seen in the alveolar spaces).

From these experimental studies we conclude that beginning patchy aeration occurs in the human infant lung when a positive pressure of 30 cm. of water is applied over a 0.2 second interval.[11] Uniform expansion is achieved (with the lungs removed from the thoracic cage) at 50 cm. (Fig. 11).

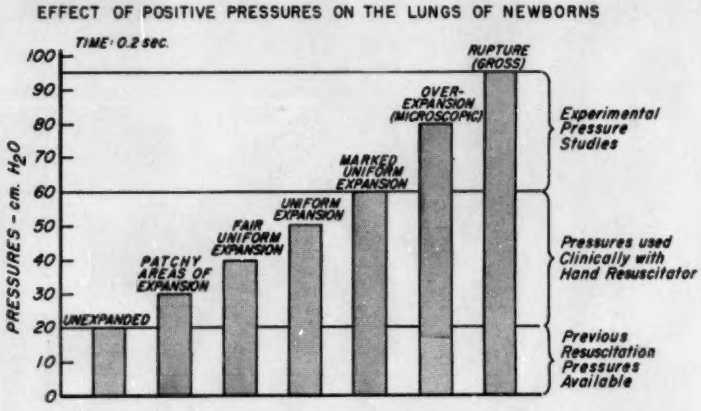


Fig. 11.—Effect of positive pressures on lungs of newborns.

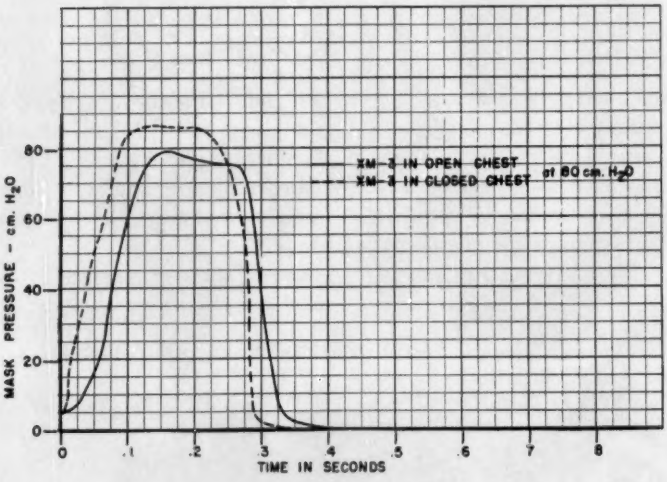


Fig. 12.—Pressure tracings at 80 cm. in open and closed chest in six-month premature twins.

Repeated studies on autopsied lungs show that a newborn's lungs are remarkably elastic (even in the open chest) after initial expansion. Contrary to a common opinion, the elastic properties of the newborn's lungs, once expanded, indicate that no negative pressure is necessary to effect expiration. The resistance offered by certain structures of the upper respiratory passageways, the thoracic cage, the abdominal viscera, etc. necessitates the use of slightly higher pressures to achieve

similar expansion when that pressure is applied via a facepiece. In laboratory studies it was shown that pressures of 5 to 7 cm. were required to overcome the upper respiratory resistance factors. The resistance and inertia offered by the chest wall causes another 5 to 7 cm. rise in the pressure over that used when the chest wall is open (Fig. 12). The optimal pressure required to achieve uniform expansion of the lungs of the newborn in the intact chest remains to be answered. Clinically, pressures of 30 to 60 cm. have been employed without damaging the lungs.

3. Respiratory-Ventilation Studies

The time and pressure relationships have been stressed in the expansion of the newborn lung. Other factors of importance are (*a*) the amount of air the infant inspires (tidal volume), (*b*) the proportion of total lung volume that is expansible, (*c*) the elastic forces of the thoracic cage, including the diaphragm and respiratory muscles, as contrasted to the elastic forces of the lung, and (*d*) the actual respiratory pattern of the newborn. In preliminary studies of the tidal volume, a thin barium solution was instilled into infant's lungs at postmortem examination to determine the amount of solution which was required to fill the infant's lungs. Figure 13 shows the extent of filling achieved with 1, 3, and 11 cc. of a thin barium solution in a 3 lb. premature infant; 11 cc. filled this infant's lungs completely. Such studies offered a rough estimate of the tidal volumes of premature and full-term infants. To study the amount of lung expanded as well as the uniformity of expansion, a fine radiopaque aerosol was introduced via the GBL Infant Hand resuscitator.* Figure 14 demonstrates that uniform and complete expansion can be achieved in a 3 lb. premature infant with 50 cm. pressure. To achieve such uniform and complete expansion, it appears that high pressures must be used initially over safe time periods, rather than hesitant lower pressures which more often give incomplete and nonuniform expansion. Following initial expansion, the pressures must then be reduced to avoid damage to the already expanded lung. To determine the relationship of the elastic forces of the thoracic cage, including the diaphragm and respiratory muscles and the elastic forces of the lung itself, a series of compliance studies was carried out. (Compliance is the relationship of change in volume to change in pressure, or $\frac{\Delta V}{\Delta P}$.) In Figure 15 the introduction of 50 cc. of air into the lungs of a 4 lb. premature infant results in the creation of a positive water pressure of 40 cm. This shows the amazing elastic qualities of the newborn lung, in that with such a pressure the lungs expand well and are elastic enough to permit the introduction of two to three times the amount of air which the infant normally inspires (tidal volume). This is another margin of safety in the use of positive pressure in the expansion of the newborn lung. Studies are under way to determine the intrathoracic pressures which the infant achieves in respiration, as well as those static forces previously mentioned which must be overcome in the initial expansion of the newborn lung.

More accurate estimates of the tidal volume were determined by means of pneumotachographic studies in live infants. These studies also furnished the important respiratory pattern of newborns and indicated not only the tidal volumes, but the respiratory rates, time of inspiration and expiration, and respiratory minute volumes. Such tracings offered a pattern of time, volume, and rhythm to be duplicated with artificial initiation of respiration in the newborn. Figure 16 gives preliminary figures on the tidal volumes of premature and full-term infants.

* Developed by V. Ray Bennett and Associates, Inc., 320 S. Robertson Blvd., Los Angeles.

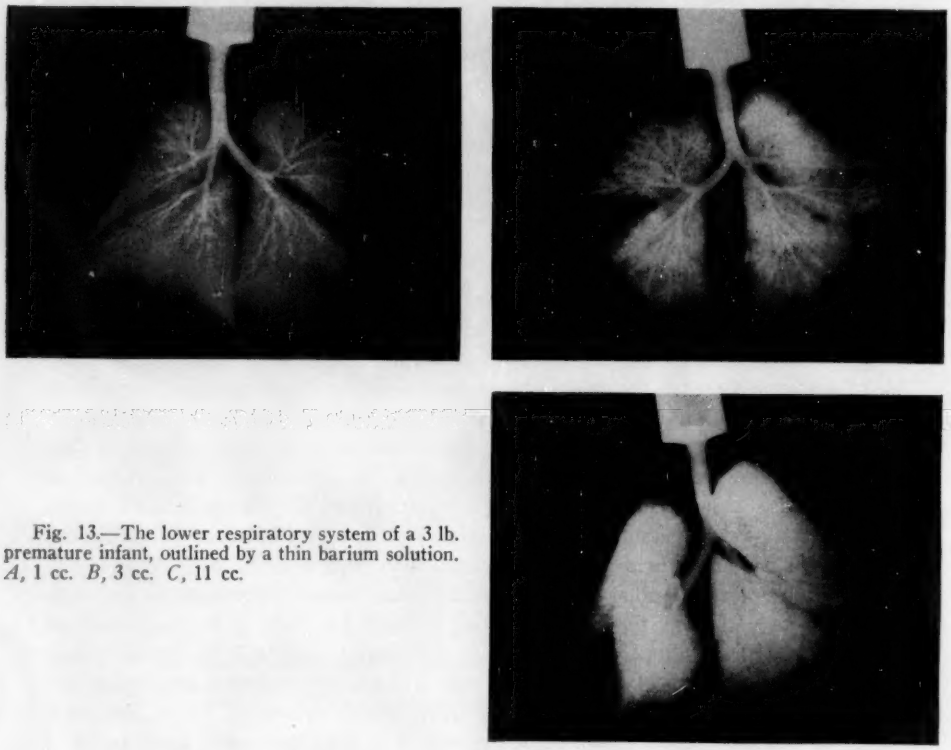


Fig. 13.—The lower respiratory system of a 3 lb. premature infant, outlined by a thin barium solution. *A*, 1 cc. *B*, 3 cc. *C*, 11 cc.

Fig. 15.—Compliance of the lungs and chest of a 4 lb. premature infant. ➡

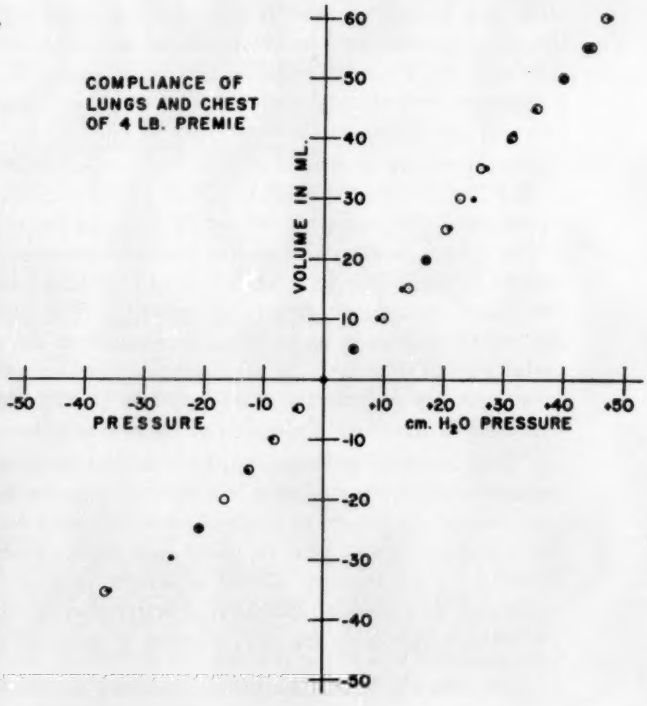
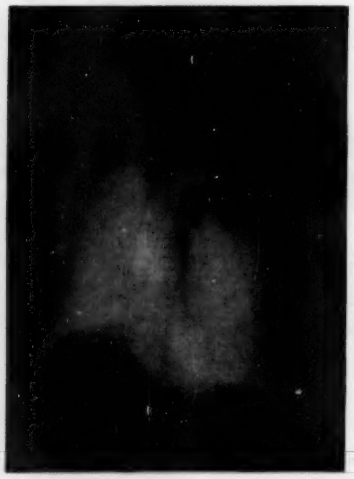


Fig. 14.—Amount and uniformity of expansion achieved in 3 lb. premature infant with 50 cm. as demonstrated by means of a fine radiopaque aerosol.



C. Clinical Studies

1. Resuscitation and Postresuscitative Care

Clinically, these laboratory findings were applied using the type of pattern most closely simulating the infant's respirations and employing pressures necessary to expand the nonexpanded or partially atelectatic lung of the newborn. Certain principles, however, must be followed in any resuscitation of the newborn. Those which the Foundation has set up are as follows:

a. Establish a patent airway
b. Promote adequate drainage of UR tract
   (1) Postural drainage
   (2) Gentle nasal and postpharyngeal suction
c. Expand lungs by intermittent positive pressure
d. Administer oxygen
e. Maintain temperature and humidity
f. Perform gastric lavage when indicated
g. Administer stimulants when indicated
   (1) Chemical (nalorphine [Nalline], caffeine and sodium benzoate)
   (2) Cutaneous or proprioceptive

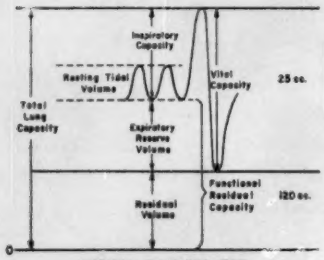
The GBL Infant Hand Resuscitator proved to be simple and available in clinical use (Fig. 17). It has proved to be the most effective resuscitator studied to date in managing the three categories of resuscitation stressed earlier.

Note the pad under the shoulders of the infant in the demonstration photo (Fig. 17). This is to insure hyperextension of the head to better establish an airway. Even more extension might be desirable.

Figure 18*A* demonstrates inadequate aeration in a full-term baby who did not breathe well spontaneously, as contrasted to Figure 18*B*, the x-ray picture some 20 minutes later, following resuscitation at 40 cm. of intermittent positive pressure via the GBL Infant Hand Resuscitator. Following resuscitation, it is important to continue observation and care of the infant. Such postresuscitative measures include the following:

a. Continue principles of resuscitation as indicated (postural drainage, intermittent positive pressure, oxygen, temperature, stimulants)
b. Maintain high humidity through water fog or mist incorporating detergents and aerosols (Mist-O_2-Gen)
c. Avoid regurgitation and aspiration by
   (1) Nothing by mouth
   (2) Hydration through hypodermoclysis and aerosols
d. Prevent infection (IM and aerosol medications)
e. Prevent hemorrhage (IM vitamin K)
f. Conserve infant's energy (minimum handling)
g. Gradually adjust to normal infant routine

Figure 19 shows a 3½ lb. (1,590 gm.) premature infant after resuscitation, receiving these measures in an Isolette and with the use of Mist-O_2-Gen therapy.



RELATIONSHIP OF TIME, PRESSURE AND TIDAL VOLUME IN PREMATURE AND FULL-TERM INFANTS

	PREMATURE	FULL-TERM
Respiratory Rate (per minute)	50-70	40-60
Tidal Volume (inspiratory in cc. per breath)		
Average	5-17	18-25
Peak	25-50	40-50
Time of Inspiration (in seconds)	0/3-0/6	0/5-0/6
Respiratory Minute Volume (cc. per minute)	300-1000	700-1500
Pressure Exerted in Respiration (positive in cm. of H_2O)		
Average	13-30	14-28
Peak	30-50	20-46

Fig. 16.—Relationship of time, pressure, and tidal volume in premature and full-term infants.

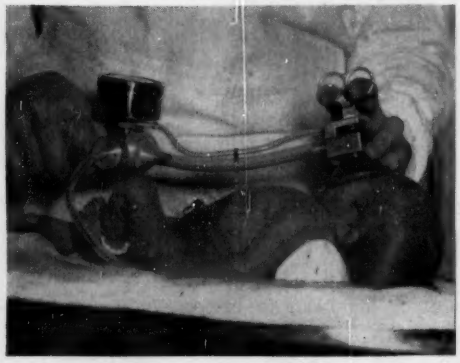


Fig. 17.—XM-4 model in clinical use.

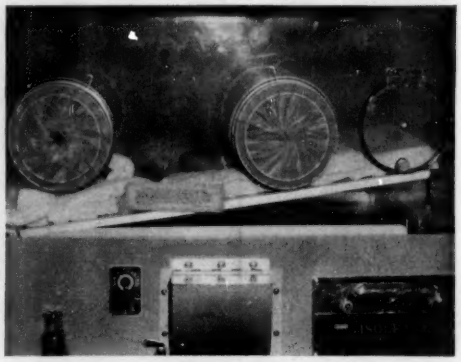


Fig. 19.—Resuscitated premature infant receiving postresuscitative measures.

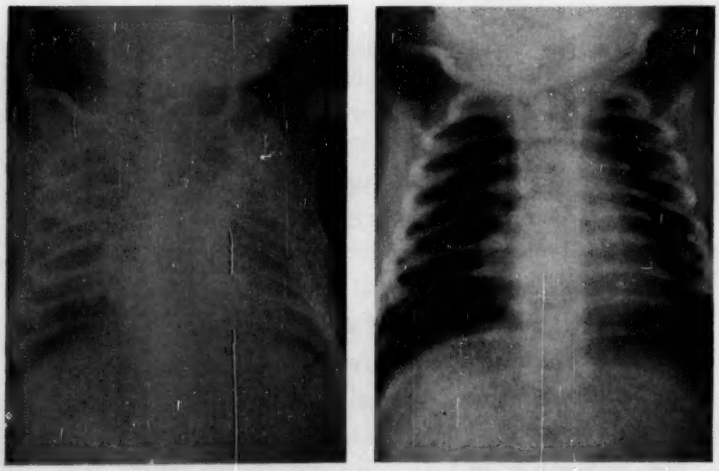


Fig. 18.—X-rays of lungs of baby. *A*, before resuscitation. *B*, after resuscitation.

2. Problems Arising in Positive Pressure Resuscitation

It is felt by many that air entering the gastrointestinal tract may interfere with diaphragmatic action. The pressures applied in these studies did not prove to be a problem when provision was made for deflation of the stomach.

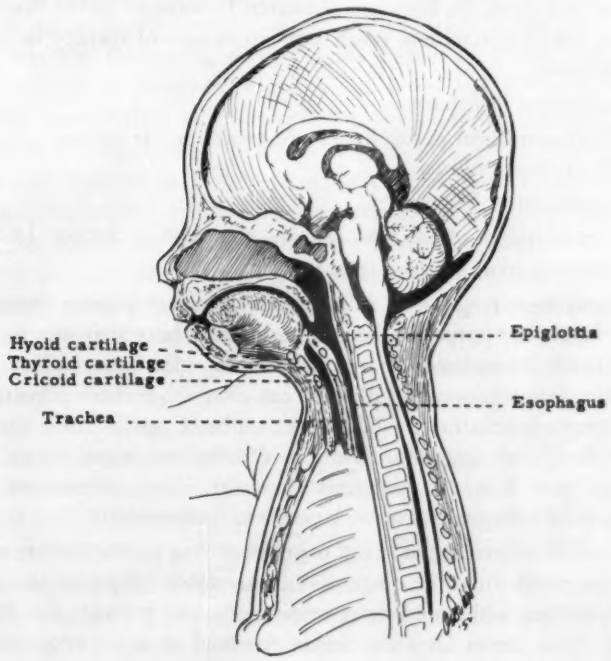
Any adverse effect of the pressure on the circulation is of a temporary nature and with application of intermittent positive pressure as described does not apparently offer a major problem. However, an expiratory time interval somewhat longer than the inspiratory phase is important to prevent undue reduction of cardiac output. If inspiration is 0.2 to 0.4 seconds, then expiration should be approximately 0.4 to 0.8 seconds, respectively.

By means of sagittal sections and moulages of the various head sizes of newborns from the fetal age of 5 to 10 months, perfection of masks to fit the various age-weight groups was made possible, as was further investigation of the problems of intubation. Figure 20 shows the difficulty one has in intubating a premature infant; in over 90% of clinical applications sufficient expansion was achieved with the facepiece to avert intubation and possible damage to the upper respiratory tract.

3. Clinical Statistics

Clinical statistics of the Foundation are rather limited. In a two-year period (July, 1952-June, 1954) 73 babies out of 975 attended warranted resuscitation. Of these, 59 or 81%, survived. Of the 14 deaths, all were in premature infants, and if correction is made for extrapulmonary deaths due to intracranial hemorrhage and congenital cardia, the survival rate rises to 90%. This appears to be a high survival rate and suggests efficiency of the hand resuscitator, but inasmuch as the hospital in which the majority of these studies was conducted has no previous obstetrical figures for comparison, further clinical evaluation seems advisable before

Fig. 20.—Sagittal section of a premature infant (1,160 gm.).



recommending the general use of the GBL Infant Hand Resuscitator. During the past nine months, six other medical centers in the United States have been participating in clinical studies with the GBL Infant Hand Resuscitator. They are as follows:

a. Sloan Hospital for Women and Babies Hospital, Columbia-Presbyterian Medical Center, New York
b. Boston Lying-in Hospital and Children's Medical Center, Harvard Medical School, Boston
c. Chicago Lying-In Hospital of the University of Chicago, Chicago
d. Mayo Clinic and Mayo Foundation for Medical Education and Research, Rochester, Minn.
e. Los Angeles County Hospital, University of Southern California School of Medicine, Los Angeles
f. Children's Hospital of the East Bay, Oakland, Calif.

Many criticisms and suggestions have been offered which have helped in the improvement of the resuscitator. Significant clinical statistics are available at this time from the obstetrical and pediatric services of the Los Angeles County Hospital. While their results are not as favorable as those of the Foundation, they report 60% survivals in infants in which the GBL Infant Hand Resuscitator was used by many different operators, including obstetrical and pediatric residents. The conclusions of Dr. Hodgman and Dr. Martini, chief investigators at the Los Angeles County Hospital, are (1) the GBL Infant Hand Resuscitator presents a sound approach to the problem of infant resuscitation; (2) it is a life-saving instrument, easy and safe to use; (3) any danger in its use is due to associated use of tracheal catheters, and (4) further controlled studies are advocated.

II. Intermittent Positive-Pressure-Aerosol Therapy in Pediatrics

During the past year, the Pediatric Research Laboratory of the Foundation has broadened the use of intermittent positive-pressure-aerosol therapy in pediatrics to include the following:

A. Resuscitation of Infants
B. IPPB (intermittent positive-pressure breathing) in infants
C. IPPB in older children

A. Resuscitation of Infants

1. Resuscitation of the newborn (as presented in Section I)
2. Resuscitation of older infants

With the excellent response of infants to intermittent positive pressure during the neonatal period for purposes of resuscitation, the next step was the use of the GBL Infant Hand Resuscitator for resuscitation in older infants. This was accomplished by using lower pressures (10 to 30 cm. of positive water pressure) in older infants requiring resuscitation (4 days to 4 months of age). These cases included assistance to those with congenital anomalies (tracheoesophageal fistula, incomplete tracheal rings [see below]), peripheral vascular shock, pneumonia, segmental atelectasis, adrenal collapse, and acute laryngotracheobronchitis.

Baby JC was first seen at the age of 6 weeks, during a consultation with another pediatrician, at which time the diagnosis of amyotonia congenita was established. Abdominal breathing with rapid short respirations was present; the lungs seemed well aerated. Two weeks later the infant returned in acute respiratory distress

with incomplete pulmonary expansion on the right, subnormal temperature, and extreme pallor and listlessness. The infant was admitted to the hospital, placed in a croupette with oxygen and aerosol consisting of a mixture of one-third Alevaire (alkylaryl polyether alcohol detergent), two-thirds distilled water, and 1 cc. of isopropylarterenol (Isuprel) delivered by means of the Mist-O_2-Gen. The film on admission (Fig. 21*A*) revealed complete collapse of the right lung with mediastinal shift. Course was rapidy downhill, and at 12 hours it was decided to apply positive pressure. Forty centimeters of intermittent positive pressure was given via the GBL Infant Hand Resuscitator over a 20-minute period with dramatic clinical improvement and x-ray evidence of partial expansion of the right lower lobe. At 36 hours another 10 minutes of intermittent positive pressure at 30 cm. was administered via the hand resuscitator with further clinical and x-ray improvement. This was repeated at 48 hours at 20 cm. intermittent positive pressure for 10 minutes with what appeared to be complete aeration clinically and x-ray expansion as shown in Figure 21*B*. The infant was discharged on the eighth day, at which time x-rays showed all but a small portion of the right upper lobe to be well expanded, with the mediastinal contents in the midline. A 17-day follow-up film showed complete expansion of both lung fields. Similar expansion of an atelectatic lung was achieved in a postoperative atelectasis in the surgical repair of a tracheo-esophageal fistula and expansion and alleviation of symptoms in an 18-day-old infant with segmental atelectasis.

Using a large hand bulb, the GBL Infant Hand Resuscitator was employed for resuscitation of a 6-year-old boy with triple fractures of the skull and in peripheral vascular shock over a two-hour period until a neurosurgeon was able to operate and decompress the intracranial bleeding (10 cm. positive pressure).

B. Intermittent Positive-Pressure-Aerosol Therapy in Infants

With the development of an attachment for the GBL Infant Hand Resuscitator which would allow the deliverance of moisture, wetting agents, and aerosols, it has almost become routine to employ aerosol therapy along with intermittent positive pressure in the treatment of infants with severe lack of respiratory exchange and pathology necessitating moisture and the dissolution of secretions (congenital anomalies, pneumonias, acute laryngotracheobronchitis, bronchiolitis, etc.).

Baby L was referred at the age of 4 days for respiratory distress and thick copious mucous secretions. Figure 22 shows sternal retraction and poor respiratory exchange. The infant was given intermittent positive pressure (40 cm.) via the hand resuscitator, combined with Tryptar aerosol therapy. During 15 minutes of treatment, rapid liquefaction of secretions occurred, and the respiratory tracings demonstrated marked change from paradoxical breathing to inflation of the lung during inspiration (Fig. 23). Treatments were given twice daily and then once daily up to and including the 10th day. The infant was recalled to the referring hospital at age 17 days, and autopsy at age 19 days revealed (1) incomplete tracheal rings; (2) small esophageal hiatus hernia, and (3) small reduplication of stomach at esophagus-cardia junction. Numerous other infants with secretions have been helped by the addition of aerosol therapy to intermittent positive pressure.

C. Intermittent Positive-Pressure-Aerosol Therapy in Children

Concurrently with the above studies in neonatal and infant respiratory complications, the Pediatric Research Laboratory was investigating the tidal volumes of older children. In studying the patterns of respiration in children with acute

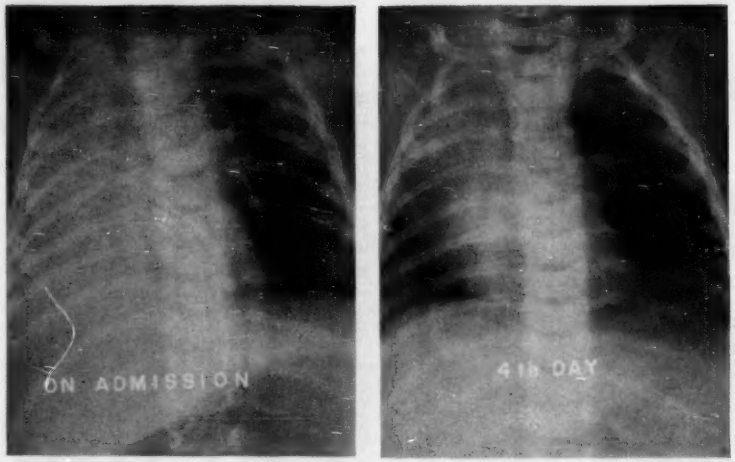


Fig. 21.—Two-month-old infant with amyotonia congenita. *A*, film on admission—atelectasis of right lung. *B*, film on fourth day, after expansion by GBL Infant Hand Resuscitator.

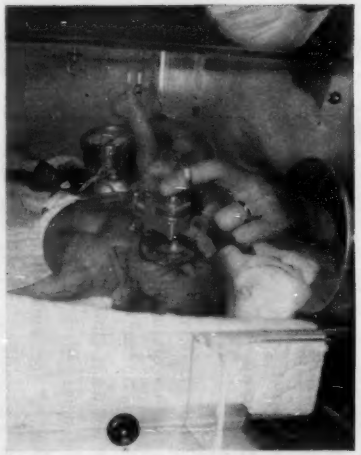


Fig. 22.—Tryptar aerosol treatment combined with intermittent positive pressure in 4-day-old infant with multiple congenital anomalies.

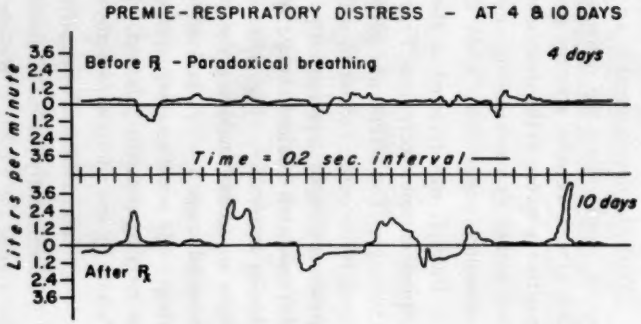


Fig. 23.—Change in respiratory pattern of 4-day-old infant with multiple congenital anomalies after Tryptar aerosol therapy.

INFANTS & CHILDREN RECEIVING INTERMITTENT POSITIVE PRESSURE-AEROSOL THERAPY (MAY 1953 TO MAY 1954)

AGE GROUP	TOTAL NO.	NO TREATED	NO OF TREATMENTS	IMPROVED E THERAPY NO.	IMPROVED E THERAPY %	NUMBER GIVEN OTHER THERAPY MED.	OTHER THERAPY SURG.	OTHER THERAPY RESUS.
INFANTS (1 DAY-1 YR.)		8	30	8	100	6	1	4
TODDLERS (1-3 YRS.)		2	6	1	50	1		
TOTAL		10*	36	9	90			
PRE-SCHOOL & SCHOOL CHILD (3-14 YRS.)								
ASTHMA	40	17	141	16	94	8		
CHRONIC COUGH	4	3	8	3	100	1		
BRONCHIECTASIS	4	4	96	4	100	2		
TOTAL		24**	245	23				
PERIPHERAL VASCULAR SHOCK		1	for 2 hrs	1	100	(Resuscitation via hand resuscitator)		

* TREATMENTS GIVEN VIA GBL INFANT HAND RESUSCITATOR
** " " " BENNETT IPPB MACHINE

Fig. 24.—Infants and children receiving intermittent positive pressure-aerosol therapy from May, 1953, to May, 1954.

and chronic types of respiratory disease, it was decided to go one step further and employ intermittent positive-pressure-aerosol therapy (using a Bennett Pressure Breathing Therapy Unit, Model TV-2P †) in some of these conditions in an attempt to improve respiratory ventilation. Experimental work by Otis, Rahn, and Fenn,[12] followed by clinical studies by Motley, Lang, and Gordon,[13] Segal and co-workers,[14] and Smart and co-workers [15] had proved not only the efficacy but the safety of this type of therapy in adults. On the basis of such studies and their success in adults, a program was set up for the use of intermittent positive-pressure-aerosol therapy in children.

1. Plan of therapy
   a. History and physical examination
   b. X-rays (chest, sinuses)
   c. Respiratory ventilation studies (older conventional methods and new pneumotachographic tracings)
   d. Allergy tests (diagnostic and therapeutic), where indicated
   e. Miscellaneous laboratory tests (CBC, eosinophile counts, sed. rate, sputum, etc.)
   f. Treatment
      (1) Intermittent positive pressure
         (a) Six to ten centimeters for age groups 3 to 6 years
         (b) Ten to fifteen centimeters for age groups 6 to 14 years
      (2) Bronchodilator agents—Aerolin (a sympathomimetic containing cyclopentamine [Clopane], isopropylarterenol [Aludrine], atropine, and procaine), isopropylarterenol (Isuprel)
      (3) Aerosols (antibiotics, detergents, trypsin [Tryptar])
2. Evaluation of therapy
   a. Symptomatic improvements (appetite, breathing, cough or wheezing, fatigability, infections, medications required, sleeping)
   b. Physical changes, (alertness, color, vigor, weight, type of respirations, change in thoracic or other configurations, breath sounds, pulse and blood pressure, temperature)
   c. Laboratory evaluations (improvement in x-rays of sinuses and chest, change in tidal volumes and respiratory patterns, improvement in miscellaneous laboratory tests)

It was felt that this type of therapy offered the following advantages:

3. Advantages of therapy
   a. More adequate ventilation of lungs with increase in minute volume
   b. More uniform alveolar aeration with improvement in blood-gas exchange
   c. Efficient means of giving oxygen or aerosol mixtures
   d. Improved bronchial drainage by high velocity expiratory gas flow
   e. More effective means for dissemination of aerosols into poorly ventilated lung areas with relief of bronchospasm and mucous plugging
   f. Effective form of deep breathing exercise

† Furnished by V. Ray Bennett and Associates, Inc., 320 S. Robertson Blvd., Los Angeles.

Children who received this therapy were those suffering from asthma (including those with various degrees of emphysema), chronic bronchitis, chronic coughs, and bronchiectasis. The number of treatments varied from 1 to 50 per child, and in a period of one year (May, 1953, to May, 1954), 245 treatments have been given without any ill effects. The results of these treatments are presented in tabular form in Figure 24.

Specific examples of three types of children treated are given below.

4. Acute asthma

TT, aged 5 years, had moderately severe asthma for over two years, with little or no relief from the usual medical therapy of asthma. His respiratory tracing was typical of the asthmatic child, with a shallow prolonged type of inspiration-expiration with very little tidal volume exchange (Fig. 25, upper tracing) during respiratory distress.

Following 10 minutes intermittent positive pressure (7 cm.) combined with an aerosol mixture of isopropylarterenol-distilled water, the clinical and respiratory improvement was dramatic, in this case, with deeper more regular pattern (increase in respiratory minute volume of 620%). This boy now combats his asthmatic attacks at home with the regular use of a Bennett Pressure Breathing Therapy Unit.

5. Chronic cough

KB, aged 11 years, had suffered from a chronic cough for two years. All medical therapy had failed. Tidal volume tracings showed frequent shallow type of respiration before treatment. After four 10-minute periods of therapy combining intermittent positive pressure (12 cm.) and an aerosol mixture of isopropylarterenol-oxytetracycline (Terramycin) and distilled water, the boy's respirations became deeper and slower. Clinically, his cough disappeared, his appetite improved, he gained weight, and in 10 months' time he has had no recurrence.

6. Bronchiectasis

CC, aged 5 years, had suffered from repeated respiratory infections from early infancy, requiring confinement to the hospital or at home in bed for long periods with high fever and little response to the usual antibiotics. Her family brought her from Kansas City to New Mexico to see if the climate would help. Physical examination: pale, T. 101 F., dry nonproductive cough, poor respiratory exchange (Fig. 27, upper tracing). Chest x-rays (Fig. 28*A*) showed poor pulmonary expansion and increased bronchovascular markings. Intensive therapy with intermittent positive pressure and an aerosol of isopropylarterenol-oxytetracycline and distilled water was given initially twice daily, then daily, q. 2 d., then q. 3 d., and finally biweekly and once a week. Criteria of improvement: (1) symptomatic (disappearance of cough and fatigue, increased appetite, able to sleep), (2) physical (disappearance of fever, return of color and vigor, clearing of chest, weight gain), (3) improvement in respiratory exchange (Fig. 27, lower tracing), and (4) clearing of x-rays (Fig. 28*B*). Improvement has continued during past three months.

7. The place of intermittent positive-pressure therapy in pediatrics

Here, as in the work of the Foundation in infant resuscitation, the statistics are few in number. More must be done to prove the efficacy and safety of the method of treatment in children. From the table presented in Figure 24, it can be seen that 24 children have received 245 treatments without any adverse effects

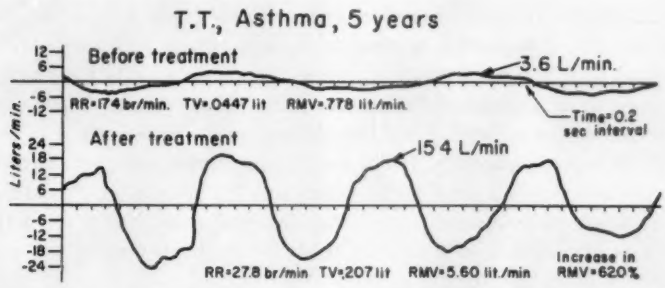


Fig. 25.—Respiratory ventilation tracing of TT, aged 5 years, acute asthma, showing response to IPPB therapy.

Fig. 26.—Intermittent positive pressure aerosol therapy being administered to 5-year-old bronchiectasis patient with Bennett Pressure Breathing Therapy Unit.

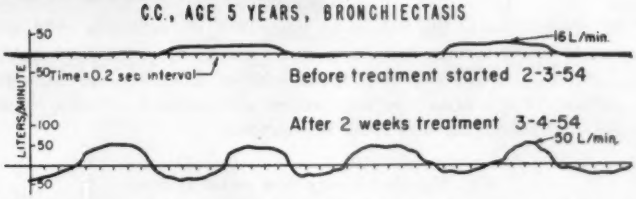


Fig. 27.—Respiratory ventilation tracing of CC, aged 5 years, bronchiectasis, showing response to intensive two weeks IPPB therapy.

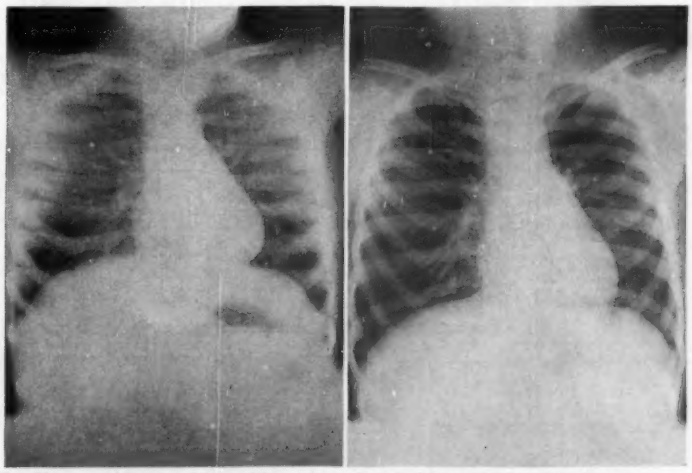


Fig. 28.—X-rays of lungs of bronchiectasis patient before and after IPPB therapy. *A*, x-rays before therapy started. *B*, x-rays after two weeks therapy.

and that 23 of these children benefited from the treatment.‡ Asthma in children can be serious and, if neglected, can lead to chronic asthma with the poorer response seen in adults to this type of treatment (Fig. 36). Whether IPPB therapy should be used chiefly as an adjunctive method of therapy in asthma in children or whether it offers a cure in the sense that the normal condition of the lung may be restored remains to be seen. Apparently, IPPB therapy in chronic coughs does offer a cure, and it goes far toward a cure in bronchiectasis. However, these are only preliminary results, and continued observation on these same and other patients over a further time period will be necessary to draw conclusions as to the permanency of the benefits of the treatment.

III. Biophysical Developments in Positive-Pressure Therapy in Pediatrics

A. Pneumotachographic Studies

To confirm the crude tidal volume measurements made by barium solution instillations into the lungs and to allow study of the true respiratory patterns of infants, the Foundation made a sensitive low-resistance orifice, (Fig. 29) similar to the Silverman Pneumotachograph principle. The orifice consists of a fine mesh screen through which the infant breathes (Fig. 30*A*). The pressure drop across the element is metered by a very sensitive pressure pick-up (Fig. 37) and hence is easily transcribed on a brush recorder. Volume flow is proportional to the area above or below the air flow curve, since the calibration is linear and may be measured with a planimeter. With this sensitive flow meter and pressure pick-up system it is then possible to determine (1) tidal volume (the amount of air an infant inspires); (2) the true respiratory pattern of the newborn (including shape

‡ Since the presentation of this exhibit in June, 1954, the asthmatic child who previously failed to respond to IPPB therapy has shown considerable improvement when trypsin aerosol was incorporated into a bronchodilator-Alevaire solution and given via intermittent positive pressure. In addition, 14 new cases have been treated with successful results, bringing the total number of children treated to 38, with 100% improvement.

Fig. 29.—Respiratory flow meter system.

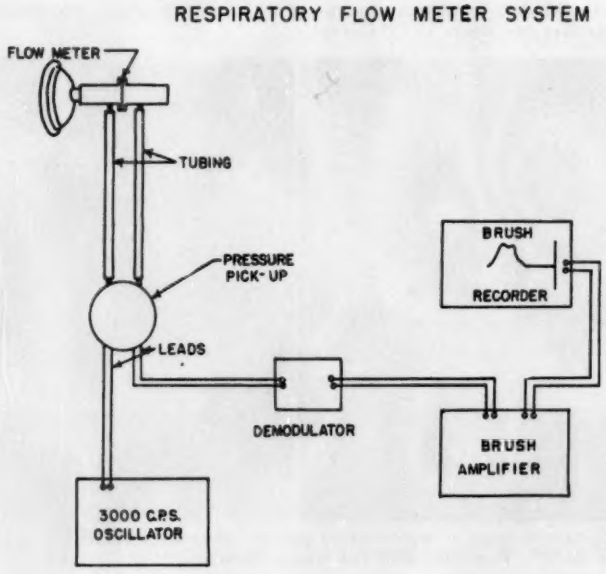


Fig. 30.—Respiratory ventilation studies being conducted with *A*, small orifice for infants, and *B*, larger orifice for children.

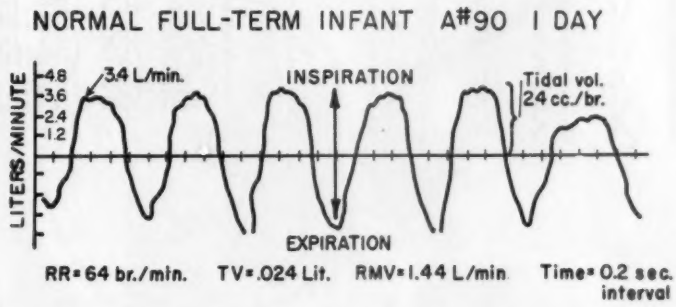


Fig. 31.—Respiratory ventilation study of normal full-term infant.

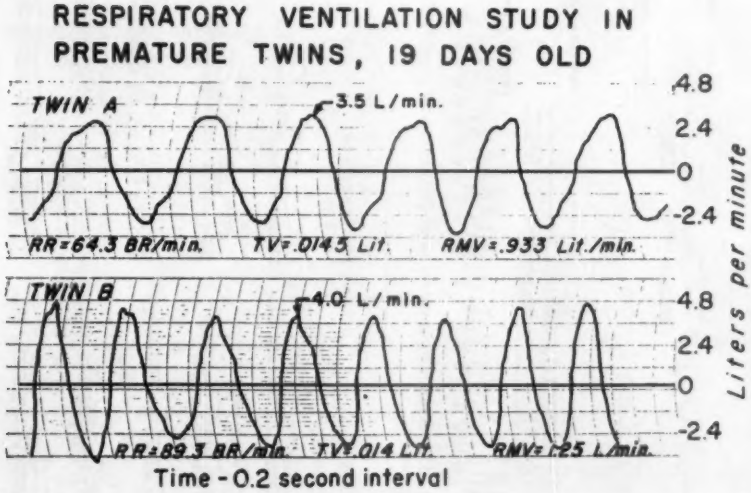


Fig. 32.—Respiratory tracings of eight-month premature twins (4 lb. 5 oz. [1,960 gm.]) at 19 days of age.

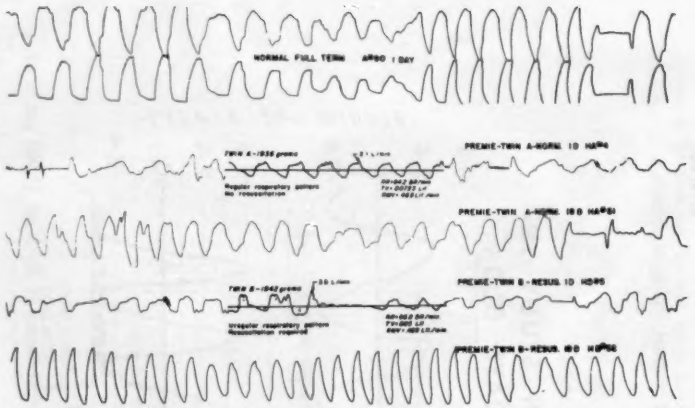


Fig. 33.—Respiratory patterns of full-term and premature infants in neonatal period.

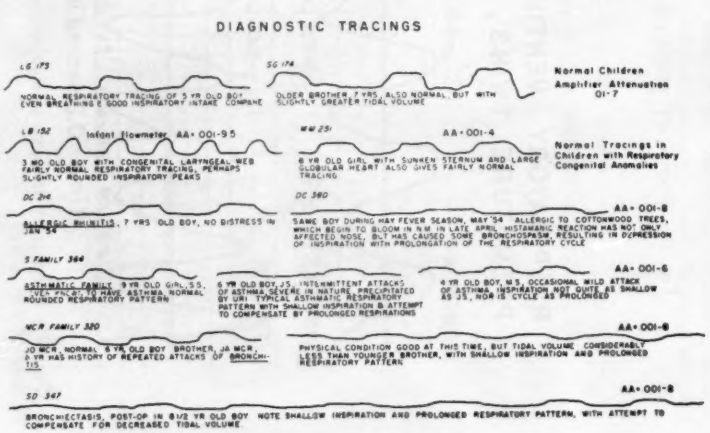

DIAGNOSTIC TRACINGS
Normal Children
Amplifier Attenuation
Infant Flowmeter
Normal Tracings in
Children with Respiratory
Congenital Anomalies
ASTHMATIC FAMILY
BRONCHIECTASIS, POST-OP IN 8½ YR OLD BOY NOTE SHALLOW INSPIRATION AND PROLONGED RESPIRATORY PATTERN, WITH ATTEMPT TO COMPENSATE FOR DECREASED TIDAL VOLUME

Fig. 35.—Diagnostic tracings in children.

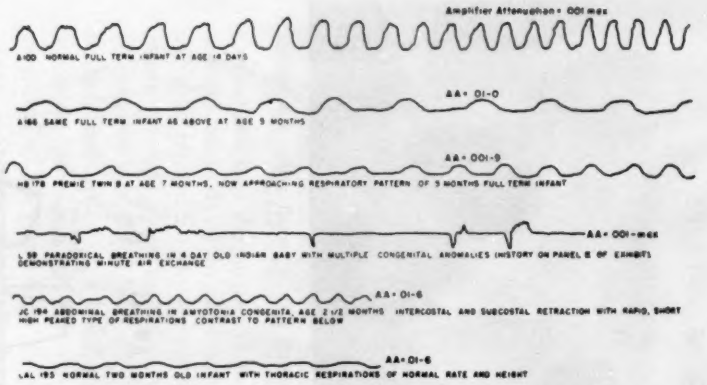

Amplifier Attenuation
A100 NORMAL FULL TERM INFANT AT AGE 10 DAYS
A166 SAME FULL TERM INFANT AS ABOVE AT AGE 9 MONTHS
HB 178 PREMIE TWIN B AT AGE 7 MONTHS, NOW APPROACHING RESPIRATORY PATTERN OF 9 MONTHS FULL TERM INFANT
L 58 PARADOXICAL BREATHING IN 4 DAY OLD INDIAN BABY WITH MULTIPLE CONGENITAL ANOMALIES (HISTORY ON PANEL II OF EXHIBIT)
DEMONSTRATING MINUTE AIR EXCHANGE
JC 194 ABDOMINAL BREATHING IN AMYOTONIA CONGENITA, AGE 2 1/2 MONTHS INTERCOSTAL AND SUBCOSTAL RETRACTION WITH RAPID, SHORT
HIGH PEAKED TYPE OF RESPIRATIONS CONTRAST TO PATTERN BELOW
LAL 195 NORMAL TWO MONTHS OLD INFANT WITH THORACIC RESPIRATIONS OF NORMAL RATE AND HEIGHT

Fig. 34.—Respiratory patterns of full-term and premature infants and other infants with respiratory distress at varying ages.

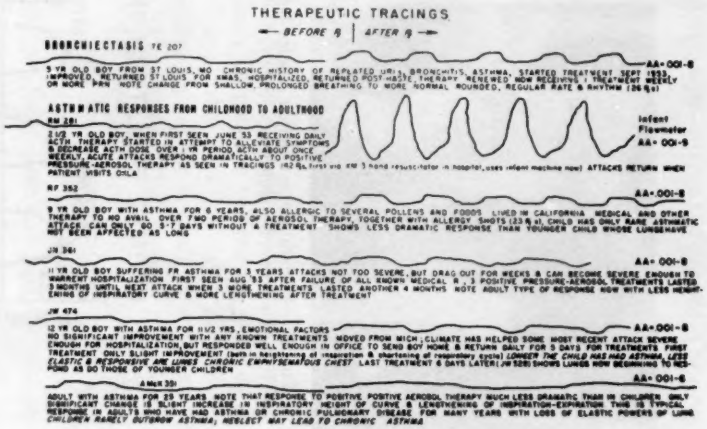


Fig. 36.—Therapeutic tracings in children.

or types of respiration, time over which inspiration and expiration occur, changes which occur with crying, external stimuli, and aging), and (3) respiratory minute volumes, with peak inspiratory volumes. It was found that a normal full-term infant at the age of one day breathed at the rate of 64 breaths per minute, with a peak volume of 3.4 liters per minute, a tidal volume (Fig. 31) of 24 cc. per breath and a respiratory minute volume of 1.44 liters per minute.

In contrast, the premature infant usually breathed at a faster rate (60 to 80), with a peak volume of 2.1 liters per minute, a tidal volume of 5 to 17 cc. per breath and a respiratory minute volume approximating 0.5 liters per minute. The premature infant usually breathes with several sharp peaked inspiratory gasps before expiration and may exhibit periods of apnea. Contrast the respiratory pattern of a set of twins (Fig. 32), the first of whom had no difficulty in starting his own respirations and at 19 days of age was approaching the full-term infant's respiratory pattern at birth. Twin B, who required resuscitation and had a very sporadic

Fig. 37.—The Clark TA-15 Pressure Pick-Up.

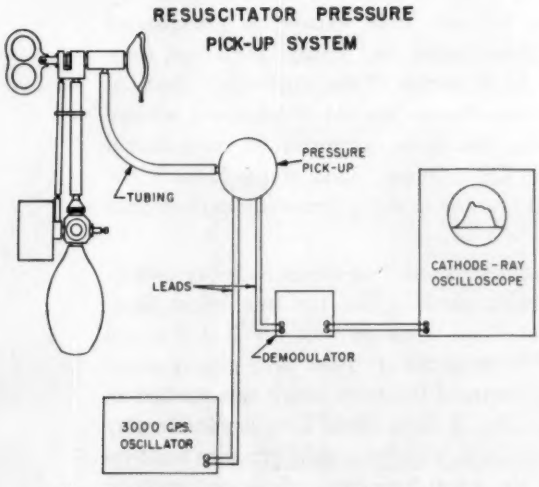


Fig. 38.—Resuscitator pressure pick-up system.

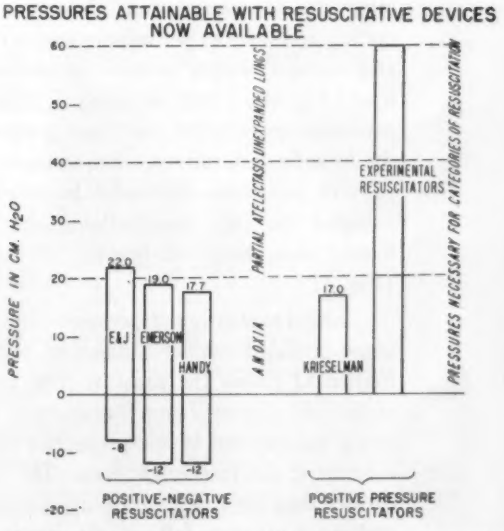


Fig. 39.—Pressures attainable with popular resuscitative devices.

initial respiratory pattern, still had sharp peaked inspiratory gasps with a high respiratory rate and did not approach the normal full-term respiratory pattern until some months later.

Other respiratory patterns of infants are shown in Figures 33 and 34.

Larger-flow sensitive orifices have been developed for use in pneumotachographic studies in older children (Fig. 30*B*). Tracings made with the use of these orifices appear in Figures 35 and 36. With the use of this respiratory flow meter system, the Foundation has been able to conduct respiratory ventilation studies in infants and children which allow both diagnostic and therapeutic evaluations of pulmonary function in normal as well as abnormal conditions in pediatrics.

B. Pressure-Time Studies

To study the actual pressures which are transmitted into the respiratory system of the infant, the Clark Pressure Pick-Up [16] was incorporated into the laboratory resuscitation system. This pressure pick-up is based on the principle of the differential transformer. A magnetic flux-carrying diaphragm is clamped between two pole pieces located centrally in two Invar steel shells (Fig. 37).

About each pole piece is a primary and a secondary coil, the former being energized by a 3,000 cps oscillator. Pressure differences across the diaphragm change the air gaps between the pole pieces and the diaphragm and hence control the output signal that is taken from the secondary winding. The resuscitator to be tested is connected to the pressure pick-up, whose output is fed through a demodulator into a cathode-ray oscilloscope which can be observed or photographed (Fig. 38).

With this system, the pressure-time relationships of presently available resuscitative devices was studied. Positive-negative resuscitators which gave positive pressures ranging from 17.7 to 22.0 cm. and negative pressures ranging from 8 to 12.0 cm. fail to expand the unexpanded lung of the newborn or correct partial atelectasis (Fig. 39). Similarly the Kriesleman, which delivers a maximum positive pressure of 17 cm. of water, does not achieve pressure necessary for these two categories of resuscitation. Nor does the Bloxsom Oxygen-Air Pressure Lock, cycling at 1.3 times per minute and exerting pressure equally to the intrapulmonary and extrapulmonary systems at precisely the same time, expand an unexpanded lung (Fig. 40). And yet many of these resuscitators may damage the lungs, since pressures are exerted over time periods far in excess of the inspiratory phase of the infant's respirations. Experimental resuscitators studied which could achieve positive pressures successful in managing the three categories of resuscitation included the Day Impulsuflator and the GBL Infant Hand Resuscitator. The former was discarded because of bulkiness and poor engineering performance (Fig. 41).

Careful evaluation of pressure-time tracings recorded on the cathode-ray oscilloscope revealed the inadequacy of tests with anything but the true infant lung. Figure 42 shows the pressure-time relationships achieved with the XM-3 model of the GBL Infant Hand Resuscitator when an artificial rubber lung closely simulating volume and elastic properties of the normal full-term infant was studied in contrast to the true infant lung. The elasticity of the artificial lung is considerably greater than the true infant lung and results in an erroneous slow pressure build-up and rapid pressure fall-off. In contrast, the infant lung responds more evenly to positive pressure and, as pointed out in Figure 12, the resistance offered by the

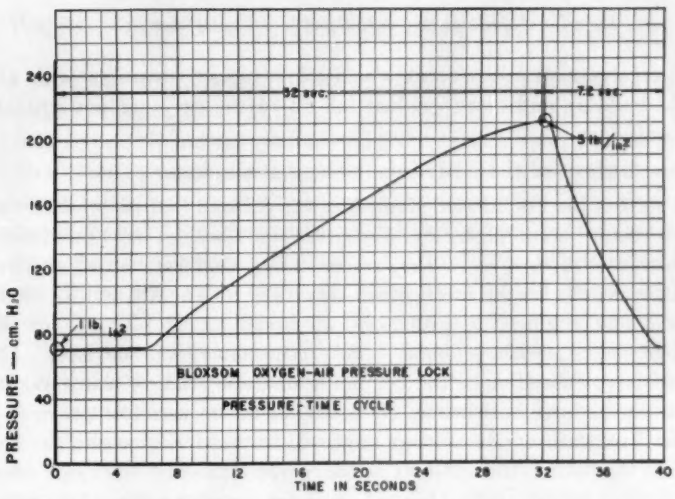


Fig. 40.—Pressure-time cycle of Bloxsom Oxygen-Air Pressure Lock.

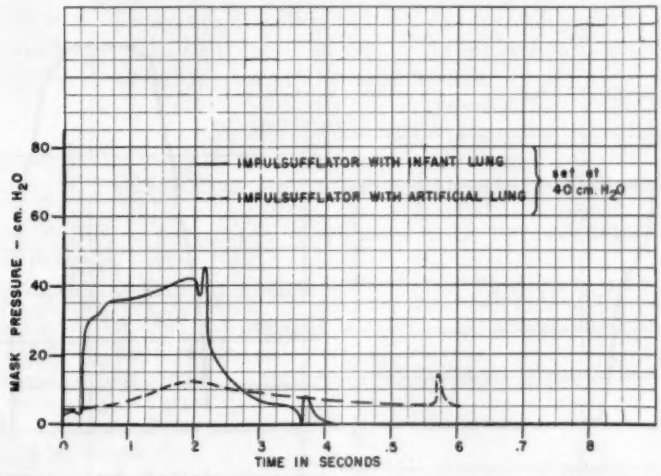

MASK PRESSURE - cm. H₂O
TIME IN SECONDS
IMPULSUFFLATOR WITH INFANT LUNG
IMPULSUFFLATOR WITH ARTIFICIAL LUNG
set at 40 cm. H₂O

Fig. 41.—Pressure-time relationships achived with Day Impulsuflator in artificial and infant lungs.

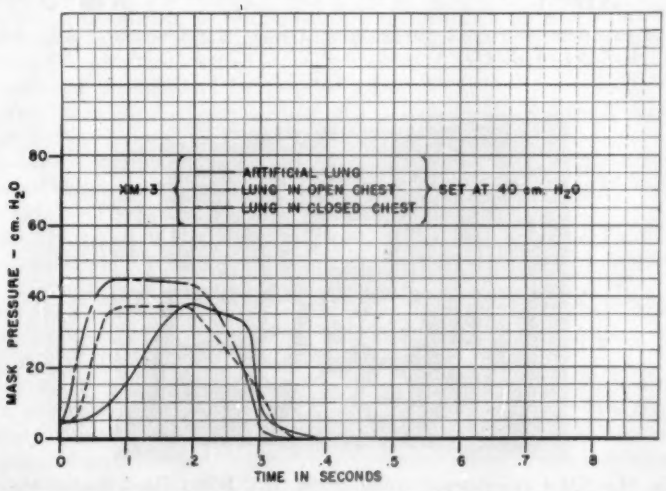


Fig. 42.—Pressure-time relationships achieved with XM-3 model of GBL Infant Hand Resuscitator in artificial and infant lungs.

chest wall creates restrictive forces which allow higher pressures with a slightly more rapid build-up and more gradual fall-off of the pressure pattern than with the chest wall open.

C. Development of the GBL Infant Hand Resuscitator

At the start of the Foundation studies, it was realized that no presently available resuscitator could accomplish the objectives outlined. The Foundation team, together with one of us (V. R. B.), as consulting engineer, set about developing a resuscitator which might meet these criteria. In an attempt to meet sound physiological and engineering principles, an initial model (XM-1) of the GBL (Goddard-Bennett-Lovelace) infant hand resuscitator was devised. Over the months that have ensued since the beginning of these studies, many modifications and improvements have been made. By means of the resuscitator pressure pick-up system, the Foundation was able to improve, from an engineering as well as a physiological standpoint, the constant pressure overload valve from one which gave a peaked humped fall-off curve (Fig. 43) to a far superior valve which now allows

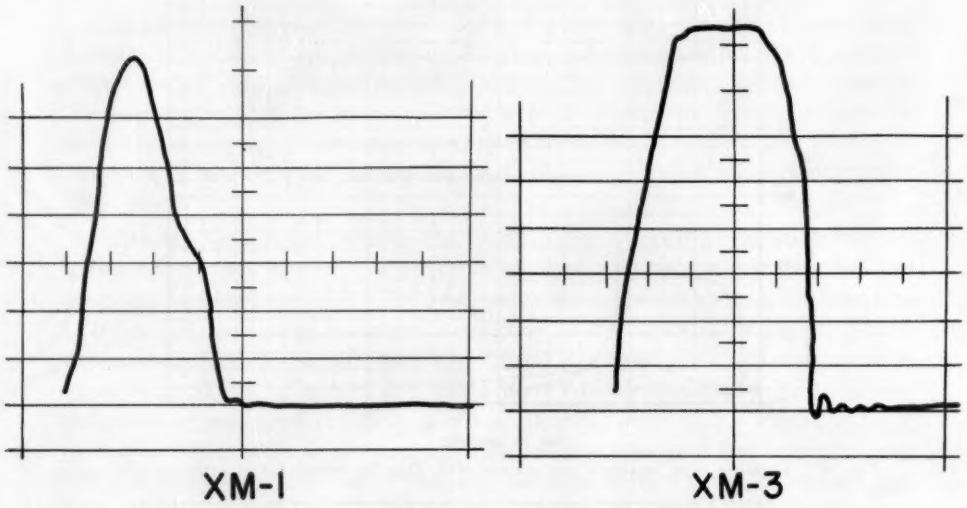


Fig. 43.—Comparison of pressure patterns of XM-1 and XM-3 models of GBL Infant Hand Resuscitator. *A*, XM-1. *B*, XM-3.

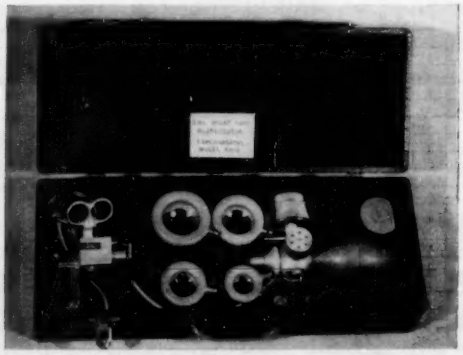


Fig. 44.—XM-4 experimental model of the GBL Infant Hand Resuscitator.

a straight up-across-down pressure pattern, which closely simulates the normal infant respiratory pattern (Fig. 31).

The many criticisms and suggestions offered by others interested in and studying the resuscitator in other medical centers around the country have contributed further to many safety features and mechanical improvements. The XM-4 model has been improved to meet the constructive suggestions and specifications resulting from all the clinical and research evaluation to date. It is believed that the GBL Infant Hand Resuscitator now meets the objectives outlined in the beginning of the program:

1. Expansion of unexpanded lungs and assistance in correction of partial atelectasis of newborns
2. Oxygenation of anoxic babies, with variation in gaseous mixtures
3. Deliverance of moisture, wetting agents, and aerosols as desired
4. Simplicity and availability

The GBL Infant Hand Resuscitator can be carried in a small case and is therefore available in delivery rooms, nurseries, pediatric services, emergency rooms, surgery, and in the physician's car (Fig. 44). It can be readily sterilized. However, it should be emphasized that it is still in the experimental stage and that further clinical evaluation and research needs to be done.

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Society Transactions

CHICAGO PEDIATRIC SOCIETY

Alfred S. Traisman, M.D., President
Regular Meeting, Jan. 19, 1954

The Postprandial Hypolipemia of Pancreatic Fibrocystic Disease: A Diagnostic Test. Dr. Edwin F. Hirsch, Dr. Alfred D. Biggs, and Dr. F. L. Phillips (by invitation).

This paper was published in full in the December, 1953, issue of the A. M. A. American Journal of Diseases of Children, page 721.

Electrophrenic Artificial Respiration in the Newborn. Dr. Lewis R. Day (by invitation), and Dr. Heyworth N. Sanford.

Thirty-seven newborn infants were treated by electrophrenic artificial respiration. Of these, 25 were treated because of apnea in the immediate postpartum period. There were 11 deaths in this group. Of these, three were considered previable. Of the remainder, five were premature infants and of 3½ lb. (1,590 gm.) or less. One had severe cerbral hemorrhage, and two showed evidence of severe prepartum asphyxia.

Twelve infants were treated for atelectasis. There were eight deaths in this group. The four survivals all weighed more than 3½ lb. It was not possible to demonstrate in this series that atelectasis in premature infants was affected by electrophrenic respiration. In full-term infants who survived, roentgenograms were taken a few hours post partum and showed more air on the treated side.

Electrophrenic respiration seems to excel other methods in certain situations. Because of its lack of versatility, it is not past the stage of experimental development, and we therefore do not recommend it for general use.

Acanthrocytosis. Dr. Meyer A Perlstein and Dr. Karl Singer (by invitation).

A new clinical syndrome is described, characterized by a genetic abnormality of the erythrocytes, which appear to be crenated or spiked. There is a history of an anemia with a sprue-like syndrome, followed in later life by a progressive neurologic involvement, characterized clinically by ataxia and tremors.

Abstracts from Current Literature

Acute Contagious Diseases

THE URINARY EXCRETION OF THE 17-KETOSTEROIDS IN ACUTE INFECTIOUS DISEASES IN CHILDREN. L. RAINERO, Minerva pediat. **6**:151 (March 15) 1954.

The amount of the 17-ketosteroids in the urine were determined in the early days of illness and again during convalescence in the cases of 41 children, 2 to 14 years of age, with diphtheria, scarlet fever, measles, or meningococcus meningitis. Some of the children in the early days of the illness showed an abnormally high secretion of the steroids; this suggested a reaction of alarm with resulting increase of secretion of pituitary hormones. These same children, later, during convalescence showed an abnormally low excretion of the ketosteroids, indicating a period of resistance or of exhaustion (Selye's adaptation syndrome).

HIGGINS, Boston.

EOSINOPHILIA IN MEASLES. G. BARTOLOZZI, S. BORGHERESI, and M. MINUCCI, Riv. clin. pediat. **53**:82 (Jan.) 1954.

Thirty-five cases of measles have been studied; a marked eosinopenia immediately resulting from the rise of the rash was noticed; this was followed by a progressive increase in the peripheral stream of eosinophile cells, which appeared to reach and to exceed the average rate in children's blood.

FROM THE AUTHORS' SUMMARY.

CARRIERS IN PERTUSSIS. M. LUST, Arch. pediat., Barcelona **32**:437 (Jan.-Feb.) 1953.

The author describes the finding of the Hemophilus pertussis bacillus in the throats of several newborn babies who had been exposed to an epidemic but never developed whooping cough. There are also carriers found in family groups where whooping cough is present in some of the children and not in others. Cultures from the throats of children not having symptoms showed the causal organism.

ALLEN, Santa Fe, N. Mex.

BILATERAL CONGENITAL CATARACTS WITH CARDIOPATHY FOLLOWING GERMAN MEASLES. ANTONIO BOSCH SALA, Rev. españ. pediat. **9**:1065 (Nov.) 1953.

Sala gives a case history of bilateral congenital cataracts with cardiopathy. The mother had German measles with an eruption June 5, and the child was born Jan. 27. Although this is later than usual for deformities, the author feels they may have been due to the infection.

A full report of examinations, x-ray, and electrocardiograms is included.

ALLEN, Santa Fe, N. Mex.

ACTIVE IMMUNIZATION IN CHILDHOOD. F. HANSEN, Deutsche med. Wchnschr. **79**:468 (March 19) 1954.

It is acknowledged that the faculty of producing antibodies increases with the age of the infant, but caution is demanded in evaluating the degree of immunity from the concentration of reagins serologically determinable. For the comparison of vaccination results it is necessary to standardize the material used for vaccination. The determination of the antigen content is regarded as insufficient and the calculation of the protective units (Schutzeinheiten) according to Prigge should be added. (This calculation is outlined in a paper by Haas, R.: *Deutsche med. Wchnschr.* **79**:43, 1954.) The following timetable for immunization is recommended: the first days after birth, BCG; at the end of the first, second, and third month, immunization with a mixture of diphtheria, tetanus, and pertussis antigens; booster immunization against diphtheria and tetanus on entrance to nursery school at the age of 3 years and on entrance to school at the age of 6 years; later, booster immunization against tetanus every three to four years and, if needed, against diphtheria; vaccination against smallpox at the age of 12 years.

AMBERG, Rochester, Minn.

THE PRACTICAL SIGNIFICANCE OF THE SERODIAGNOSIS OF EPIDEMIC PAROTITIS. OSKAR VIVELL and ROLF MARQUART, Deutsche med. Wchnschr. **79**:574 (April 9) 1954.

Attention is called to the presence of antibodies in the serum of patients with mumps. The complement fixation test is preferred using a viral antigen. In cases of mumps, the highest titer is obtained usually after the 14th day of the disease. A titer reaching 1:64 or higher is regarded as evidence of the specific infection, allowing the diagnosis even in the absence of the parotitis. If present in the mother's blood, the antibodies pass through the placenta to the fetus. The antibodies disappear from about the age of 6 to 24 months in about 80% of the cases. At the age of 14 years, the antibodies were again present in 88% of persons examined.

AMBERG, Rochester, Minn.

INCREASED FREQUENCY OF MENINGOCOCCAL DISEASES WITH WATERHOUSE-FRIDERICHSEN SYNDROME. H. U. SAUERBREI, Deutsche med. Wchnschr. **79**:785 (May 7) 1954.

Sauerbrei's paper records the occurrence of 15 menigococcal diseases in children within a period of about a half year. Of these, 13 presented the picture of the Waterhouse-Friderichsen syndrome. In the vicinity several more cases of the syndrome were observed. Of the 15 children, 11 were less than 2 years of age and 4 were 3 to 4 years old. Before publication, six further cases were seen. Eight patients were treated with high doses of penicillin, sulfonamides, and cortisone, and of these, six were cured.

AMBERG, Rochester, Minn.

A WINTER EPIDEMIC OF POLIOMYELITIS ACUTA ANTERIOR. K. BOVENTER, Med. Klin. **49**:215 (Feb. 5) 1954.

During the winter, 1951 to 1952, a small epidemic of poliomyelitis occurred in an industrial city of 24,000 inhabitants, where no cases of this disease had been observed for many years. After the first incidence, probably introduced from outside, 32 cases of poliomyelitis occurred, starting about one month later. It is of interest that extraordinarily warm and sunny weeks preceded the outbreak. An unusually high percentage of the disease (16%) occurred in more than one member of a family.

AMBERG, Rochester, Minn.

ENCEPHALITIS IN GERMAN MEASLES. P. FABIAN, Pediat. listy **8**:196, 1953.

A widespread German measles epidemic in middle Slovakia with the occurrence of neurological complications is reported. Six cases of meningoencephalitis followed a typical rash, two cases showed no preceding rash; the children were between 3½ and 13½ years old, the course of the illness varied from mild to severe; there were no deaths, and complete recovery without lasting after-effects was achieved. Apart from the neurological signs, there were often signs of marked psychological disturbance. As regards therapeutic lumbar puncture, hypertonic glucose and convalescent serum proved to be of value.

FROM THE AUTHOR'S SUMMARY.

PENICILLIN TREATMENT AND IMMUNITY TO SCARLATINA. JUSTUS STROM, Acta pædiat. **43**:267 (May) 1954.

The effect of penicillin on immunity following scarlatina has been investigated. Comparative studies were made of the incidence of second attacks in Stockholm during the prepenicillin period 1938-1946 and during the penicillin period 1947-1951. Parallel series in addition comprised treated and untreated cases.

Early second attacks, or relapses, in hospital diminished considerably due to the lesser risk of cross-infections. The frequency of early second attacks within the first three months of recovery was, however, as great as previously. Apart from the possibility of impaired immunologic conditions during this first period, the factors governing the recurrence of attack at this stage are nowadays the risk of infection from the home environment and, also, to some degree, the lack of intensity in treatment. A reduction in the early second attacks should be obtainable as a result of effective treatment and after-control of the patients and of eradication of bacteria from the environment.

As regards the late second attacks (after three months), the frequency was many times higher in 0- to 14-year-old children who had been treated with penicillin than in the untreated.

With increase in age, moreover, the relative frequency of second attacks rose still more. The interval between attacks was much shorter than in the prepenicillin period. There was, thus, a marked deterioration in immunity, which was all the more pronounced as the children grew older.

It proved probable that the risk of a new attack of scarlatina could be diminished by postponement of treatment. This is in agreement with the observation that second attacks among penicillin-treated desquamation cases are very much less numerous than among treated cases of scarlatina.

The value of penicillin in the treatment of scarlatina is so great that its use should be continued. The tendency to second attacks, even recurring attacks, is, on the other hand, so considerable, at least in built-up areas, that a modification in the treatment should be introduced. Postponed treatment can be adopted in all cases in children except those exhibiting markedly toxic symptoms or purulent complications.

FROM THE AUTHOR'S SUMMARY.

RECEPTOR-DESTROYING ENZYME IN POLIOMYELITIS. B. HOFMAN, Maandschr. kindergeneesk. **22**:153 (May) 1954.

The presence of a "receptor-destroying enzyme" (RDE) in the saliva from many people is described. It is identical with the enzyme found in filtrates from cultures of Vibrio cholerae. This enzyme has the capacity to destroy the cell receptors for viruses of the mumps-influenza group and the Columbia-SK group. Simultaneously it brings about, on red cells, the so-called Thomsen-Friedreich phenomenon.

The enzyme appears to be present more frequently and in larger quantity in normal children than in normal adults. It occurs more frequently and in a larger amount in cases of poliomyelitis than in control persons.

Some hypotheses are discussed as to the meaning of these findings. It is stressed that the presence or absence of this enzyme cannot help us in the diagnosis of poliomyelitis. For this we need the isolation of the virus from the feces or the clear establishment of a rise in antibody titer in two successive serum samples.

FROM THE AUTHOR'S SUMMARY.

Acute Infectious Diseases

ETIOLOGY OF CYTOMEGALIA INFANTUM. W. H. MINDER, Schweiz. med. Wchnschr. **83**:1180 (Dec. 5) 1953.

Material for the electron microscope optical examination was obtained from a premature newborn girl who died 14 days after birth. The pathological diagnosis was generalized cytomegalia inclusion body disease with protozoon-like cells in the pancreas, kidneys, liver, and thyroid gland. There was a splenohepatomegaly and a general hemorrhagic diathesis. On examination of these protozoon-like giant cells by means of the electron microscope, numerous globular corpuscles, mostly in aggregation, and measuring about 100 mμ were seen in the protoplasm and in the peripheral zone of the nucleus. These corpuscles are regarded as the salivary gland disease virus.

AMBERG, Rochester, Minn.

MYALGIA EPIDEMICA (BORNHOLM DISEASE, PLEURODYNIA). H. JAKOB and E. SCHWARZ, Schweiz. med. Wchnschr. **84**:640 (June 5) 1954.

Attention is called to atypical cases of myalgia epidemica on the basis of two patients about 20 years of age. From the stool of each, Coxsackie virus was cultured on the 17th day of the disease.

AMBERG, Rochester, Minn.

ORNITHOSIS IN CHILDHOOD. WERNER STROBEL, Deutsche med. Wchnschr. **78**:176 (Jan. 29) 1954.

Three patients were observed in one family, the father, a girl 8 years old, and a boy 5 years of age, in which, according to the clinical and, in the two children, the x-ray findings together with the complement-fixation titer justified the diagnosis of psittacosis. Father and son responded very well to chlortetracycline (Aureomycin); the daughter was treated with penicillin without striking results.

AMBERG, Rochester, Minn.

RESULT OF A NEW THERAPY OF SEVERE TETANUS WITH LOW MOLECULAR POLYVINYLPYRROLLIDON. RENÉ SCHUBERT, Deutsche med. Wchnschr. **78**:179 (Jan. 29) 1954.

Tetanus developed in a 14-year-old boy after an incubation period of only three days. At least six days after inability to sit up, he was admitted to the hospital with fully developed tetanus which did not respond to antitoxin treatment given for a period of five days. Then polyvinylpyrrolidon (Periston N) was given intravenously. The median molecular weight of this Kollidon preparation is about 25,000. It was given in a dosage of 100 ml. of a 6% solution for five days. From the second day, definite improvement was noted.

AMBERG, Rochester, Minn.

CAT-SCRATCH FEVER. ROBERT DEBRE and JEAN-CLAUDE JOB, Acta pædiat. **43**: Supp. 96 (March) 1954.

Following the observation, during more than 10 years, of subacute suppurating adenitides after cat scratches, Debre and Job managed, since 1947, to identify the fever with certainty with the help of the specific intradermal reaction conceived by Lee Foshay. Debre and Job described the fever first in association with M. Lamy, M. L. Jammet, L. Costil, and P. Mozziconacci, in January, 1950, under the name of cat-scratch fever (Maladie des griffes de chat). Shortly afterward, many cases were reported in France, Switzerland, the United States and a large number of other countries, thus conforming, widening, and completing our first findings. A description of the disease is given here on the basis of our 80 cases and of the whole of the published cases, the number of which is well over 300.

Cat-scratch fever is found throughout the world, in all climates, and in all age groups. A cat is the usual vector and inoculating agent; a contact with one or several cats was definitely established in 83% of the cases. In 54% the disease was inoculated by a cat scratch, and in 6% by a cat bite, whereas 13% of the patients developed a lesion around an excoriation due to a thorn, a metal or wood fragment, or insect sting. The existence of familial epidemics, at the origin of which a cat living with the family was always found, further substantiates the value of these statistical data. Nevertheless, the transmission of the disease is only incompletely known; 17% of the patients had no contact with a cat, and 27% failed to remember an inoculating trauma. It seems that the cat's transmitting capacity does not exceed several days or weeks, the cat itself not being affected. The source of the virus and its natural reservoir have not been discovered. Nevertheless, there must exist a reservoir, as suggested by our report on an endemic of 12 cases transmitted during five years by cats of a small town in the vicinity of Paris.

Although cat-scratch fever is certainly of infectious nature, the causative pathogen has not so far been identified. Mollaret, Reilly, Bastin, and Tournier have reported that the disease can be transmitted to men and monkeys, and that it may inconsistently give a complement deviation with the antigen of lymphogranuloma venereum. These authors demonstrated the existence in monkeys, and sometimes in men, of cellular inclusions bearing evidence, in their opinion, of the existence of a virus; Debre and Job failed to discover and to study these inclusions in their sections of human lymph nodes. Debre and Job do not think that it is possible to establish a full relationship with the viruses of the ornithosis-lymphogranuloma venereum group, because of morphological differences and chiefly of the failure of inoculations on embryonic eggs. The authors report the results of their investigations carried out with ganglionic pus and nodes taken, unfortunately, fairly late; the cultures on various media, the inoculations on tissue cultures, on the yolk sac of incubated eggs, and ultimately to various rodents and monkeys failed. On the contrary, the inoculation of crushed nodes in rodents treated with cortisone enabled the authors to obtain cutaneous lesions without adenopathies transmissible in series of animals; their specificity is not unquestionable.

The typical form of cat-scratch fever associates a cutaneous lesion of inoculation, a regional adenitis, and general manifestations. The duration of incubation ranges from two to six weeks. Several nodes of the same group, most often the axillary one, are involved, or sometimes two connected groups; the adenitis is of very variable volume, either solid or fluctuating; it suppurates in 50% of the cases. The inoculation lesion is to be seen in one half of the patients, under the aspect of a small macula or papule, which is often precocious and transient but may appear again when the adenitis is formed. At this stage, a slight attack of fever lasting about 10 days, is commonly seen. The course is always benign. The adenitis either disappears spontaneously or the pus is evacuated through a fistula possibly simulating tuberculosis; anyway, recovery is complete within several weeks or months, a protracted course being very uncommon.

Laboratory examinations are necessary and an unquestionable diagnosis must be based on a positive intradermal reaction. The injection of 1/10 ml. of diluted pus induces the formation, three or four days later, of a pinkish papule with a diameter of 6 mm. or more, and sometimes a focal or systemic reaction. This test is specific and lasting; but some failures require the use of various different antigens. The complement deviation test is not specific and sometimes noncontributory. Examination of sterile pus or of ganglionic smears obtained by punctures is of diagnostic value, as well as a biopsy performed in several cases. The blood count is not much modified.

On the basis of all these findings, it is possible to differentiate cat-scratch fever from other subacute adenitides. In the absence of inoculations, cutaneous lesions, adenitides due to common pyogenic organisms, tuberculous adenitis, Hodgkin's disease, and lymphogranuloma venereum are to be discarded in the first place; diagnosis with ganglionic tumors, acquired toxoplasmosis, and giant follicular lymphadenopathy is more uncommon. If there is an adenitis associated with a skin lesion, tularemia, and other Pasteurella infections, sodoku, and mycoses should be considered. If there is a polyadenitis, infectious mononucleosis, brucelloses, and sarcoidosis should be considered.

Several clinical forms of cat-scratch disease are known at present. These are symptom-free forms, protracted forms, so-called "pseudovenereal" inguinal forms, forms with superinfection of the adenitides by the tubercle bacilli. A maculous eruption or true erythema nodosum may accompany the adenitis. Especially interesting are the ectopic forms of the disease; oropharyngeal (associating tonsillitis or pharyngeal abscess with a cervical adenitis), palpebral, conjunctivitis, mesenteric (abdominal adenitis simulating appendicitis), and thoracic (with involvement of the mediastinal nodes or primary atypical pneumonia). The most special forms are those with neuromeningeal accidents; encephalitis, myelitis, neuritis, and meningitis. The number of cases in which cat-scratch fever is to be suspected and the intradermal reaction resorted to, is ever increasing. The diagnostic criteria of these forms are given with accuracy, and it is emphasized that in these forms, too, a cat is the usual carrier of virus.

The pathological examination of a cat-scratch fever adenitis usually shows several nodes of different volume, circumscribed by loose periadenitis and containing areas of suppuration of variable size. The microscopic study shows the disappearance of the normal node pattern, which is replaced by an inflammatory granuloma with areas of reticular hyperplasia with large clear cells and other areas infiltrated by round cells, polymorphonuclear cells, and some plasmocytes; the granuloma is later the seat of necrotic foci which melt into pus. Typically, there is a follicular structure; lymphoplasmocytic peripheral circle, area of epithelioid cells "en palissade" scattered with Langhans giant cells, and a necrotic center. Stress is laid on the very marked reticular and endothelial hyperplasia and on the very early appearance of sclerosis at the periphery of the granuloma. This histological pattern is not specific of cat-scratch fever and is related to aspects found in other ganglionic granulomas (tuberculosis, Hodgkin's disease, sarcoidosis, lymphogranuloma venereum, infections, and mycotic adenitides). The skin lesion of inoculation is made of a granuloma similar to that of the node, with the same reticular hyperplasia.

The action of treatment is difficult to appraise because of the usually beneficial course. A puncture of the node permits the removal of the pus and is the first treatment to carry out. Antibiotics should be used if the adenitis or fever outlasts the puncture; chlortetracycline (Aureomycin) induced the cessation of the fever and accelerated the recovery of the adenitis in nearly all treated cases. Other authors obtained similar results with oxytetracycline (Terramycin), chloramphenicol, and erythromycin. If the adenitis is not cured by antibiotics, surgical removal may be indicated. Antibiotics should be given in all ectopic or complicated forms.

FROM THE AUTHORS' SUMMARY.

Chronic Infectious Diseases

MUST WE TREAT THE INITIAL TUBERCULOSIS OF CHILDREN AND ADOLESCENTS? ROBERT DEBRÉ and H. E. BRISSAUD, Presse méd. Supp. 25, p. 523 (April 7) 1954.

The present availability of active and wieldable therapeutic products has, of course, encouraged the tendency to act as early as possible in the presence of an initial tuberculous episode. With a view to answering the question whether this tendency is or is not commendable, the authors

discuss the indications for treatment and methods of treatment in this initial stage of tuberculosis. This stage may, during the following years, be complicated by pleural, pulmonary, or extrathoracic localizations.

Most valuable work has been carried out regarding the danger which may be incurred by procrastinating in the use of the powerful antibiotic medication. The risk may be well determined in relation to the age of the patient, the degree of contamination, and the character of the initial group of persons. It is on such data that the prognosis of an initial tuberculosis in a child and in an adolescent is established and that the therapeutic indications are formulated.

The treatment to be advocated consists of concurrent administration of isoniazid and *p*-aminosalicylic acid (PAS) for a period ranging from six to eight months. Adjunction of streptomycin should be reserved for infants under 2 years of age. The beneficial effect of such a treatment upon the incidence of late tuberculous manifestations permits much hope, but this should be demonstrated by statistical inquiries similar to those which are now being carried out in France.

FROM THE AUTHORS' SUMMARY.

TREATMENT OF TUBERCULOUS MENINGITIS IN CHILDREN WITH ISONIAZID (RIMIFON). M. VEST, Ann. pædiat. **182**:158 (March) 1954.

Seven cases of tuberculous meningitis in children between the ages of 1 and 7 years are described that were treated with isoniazid (Rimifon) intrathecally and by mouth. Two children also had miliary tuberculosis. Five of them were cured with isoniazid alone. (Duration of observation 9 to 16 months.) One patient who was treated for three months only had a relapse two and one-half months after the medication was stopped, which was cured by combined treatment with streptomycin and *p*-aminosalicylic acid (PAS). The last case, a Mongoloid idiot who entered treatment rather late, did not react very well to treatment so far lasting five months.

Vest found that the most favorable dose for peroral isoniazid was 5 to 10 mg. per kilogram of body weight and 7.5 to 15 mg. for intrathecal application. Peroral treatment was carried out generally for eight to nine months, while the intrathecal therapy was given for four to six months with the first daily and later less frequent lumbar punctures. In this way grave side effects were avoided.

FROM THE AUTHOR'S SUMMARY.

TUBERCULOUS GLANDS OF THE NECK: ONCE AND NOW. W. LOFFLER, Bull. schweiz. Akad. med. Wissensch. **9**:402 (Dec.) 1953.

Tuberculous throat lymphomas, which are frequent nowadays and are caused by drinking infected milk, are a tuberculous manifestation about which we have had information for many centuries (since 1608) owing to the power ascribed to the French kings of being able to heal these glands by a touch of the hand ("toucher les ecrouelles").

The complete eradication of disease from the cattle population would, in practice, eliminate this form of tuberculosis, which is the expression of a primary alimentary infection.

FROM THE AUTHOR'S SUMMARY.

OLIGOSYMPTOMATIC TOXOPLASMOSIS. O. THALLHAMMER, Helvet. paediat. acta **9**:50 (April) 1954.

In less severe cases of congenital toxoplasmosis in any children beyond infant stage, there are no characteristic clinical or serological signs. To determine whether or not congenital toxoplasmosis plays a role in the production of congenital brain lesions, a statistical analysis must be made between the frequency of toxoplasmosis as found in normal children and children with congenital brain lesions in similar age groups. Using the Sabin-Feldman method, Thallhammer has tested 300 normal children and 266 suffering from congenital brain lesion. The results were compared by age groups. The author has found in the younger groups that asymptomatic postnatal toxoplasmosis does not yet occur (1 to 5 years of age) and that approximately 17% of the children with congenital brain lesions had been infected by toxoplasmosis. In children from 6 to 10 years old showing congenital brain lesions, the number of cases of toxoplasmosis increases at the same rate as in the normals of the same age group. In children from 11 to 14 years old showing congenital brain lesions, the number of positive cases decreases, in contrast to the increasing contamination occurring in the normal (number of deaths, reduction of antibody

production after congenital infection). The higher frequency of toxoplasmosis infection in children with congenital brain lesions is statistically significant in all age groups. Thus it seems to be proved that a form of congenital toxoplasmosis with very few symptoms exists and plays an appreciable part in the genesis of congenital brain lesions. The most frequent manifestations are oligophrenia and epilepsy.

FROM THE AUTHOR'S SUMMARY.

THE ROLE OF THE "UNKNOWN SPREADER" IN THE TUBERCULOUS PRIMARY INFECTION IN CHILDREN. H. WISSLER and M. SCHERRER, Schweiz. med. Wchnschr. **83**:1258 (Dec. 26) 1953.

The possible source of infection of 293 children with certain primary pulmonary tuberculous infection could be readily ascertained at 55%. The source of infection remained unknown in 45%. The children were infected outside of their home. The source remained unknown in 45% of children less than 5 years old.

AMBER, Rochester, Minn.

ALACTOFLAVINOSIS IN CHILDREN. G. BOI, Ann. ital. pediat. **6**:26 (Feb.) 1953.

The author reports four cases of secondary alactoflavinosis, in an 18-month-old child with pulmonary miliary tuberculosis, in a 19-month-old child with tubercular infiltrate and subchronical enteritis, in another child of the same age with hydrocephalus and enteritis, and in a 10-year-old boy with reaction of the hilum, peribronchial adenopathy without important conditions of alimentary lack of vitamin. Fully agreeing with other authors, the author thinks that in the genesis of these conditions the initial disease is very important; i. e., the tubercular factor and enteric disease with cellular alteration chiefly hepatic may prevent the utilization of lactoflavin in the same mechanism observed. Therefore these cases belong to secondary dysvitaminoses where the organic factor is very important.

FROM THE AUTHOR'S SUMMARY.

THE RELATIVE IMPORTANCE OF THE METHOD OF ADMINISTERING BCG OR OF KILLED BACTERIA IN PROPHYLACTIC VACCINATION. A. DEGLI ESPOSTI, Clin. pediat. **35**:56 (Jan.) 1953.

The observations were made with four groups of guinea pigs. The animals in the first group were given BCG intradermally, those in the second group were given BCG subcutaneously, those in the third group were given Petragnini's anatoxin intradermally, and those in the fourth group were given Petragnini's anatoxin subcutaneously.

The enzyme reaction of Sivori was determined quantitatively with each animal 10, 20, and 30 days later; these determinations indicated a delayed protective reaction in the group receiving anatuberculin subcutaneously as compared with the other groups.

HIGGINS, Boston.

THROMBOPENIC PURPURA APPEARING IN A TUBERCULOUS PATIENT UNDERGOING TREATMENT WITH ISONIAZID. L. MARTONI and B. FORTI, Clin. pediat. **35**:615 (Sept.) 1953.

A 4-year-old girl with cervical and hilar tuberculous adenitis had received penicillin, which had been followed by some lowering of the body temperature. Then, for five days, 300 mg. per kilogram of body weight of dihydrostreptomycin was given daily; this was followed by the giving of 3 mg. per kilogram of body weight of isoniazid daily for five days. The patient then received another course of the dihydrostreptomycin for four days, which was followed by a course of 7 mg. per kilogram of body weight of isoniazid. On the second day of the second isoniazid course the child developed an acutely diffuse purpura with epistaxis and bleeding from the gums; the spleen was palpable. The platelet count, which had been 230,000, was now found to be only 5,000. The medication was discontinued and transfusions were given. No new purpuric lesions were seen after eight days, and after two weeks the platelet count had risen to 200,000. The dihydrostreptomycin therapy was then resumed for 10 days; then a course of 3 to 5 mg. per kilogram of body weight of isoniazid was given for 20 days. The size of the spleen became normal, and no further purpuric lesions appeared.

The suggestion is made that the purpura may have been due to an allergic reaction caused by a sudden liberation of tubercular toxin from the action of the isoniazid.

HIGGINS, Boston.

ALLERGY TO TUBERCULIN AMONG THE ELEMENTARY SCHOOL PUPILS IN MODENA. L. COTTAFAVI, Clin. pediat. **36**:149 (Feb.) 1954.

Two thousand nine hundred school children in the city of Modena, 6 to 12 years old, were given skin tests using anatuberculin. Of these children, 25.6% had positive tests; the percentage was higher in girls (28%) than in boys (23%). The percentage among 6-year-old children was 19% and among 12-year-old children 32%. Twenty-seven per cent of the children with positive reactions had a positive family history of exposure to tuberculosis. Only a small percentage of the children with positive tests showed x-ray changes of parenchymatous changes or of calcified hilar glands.

HIGGINS, Boston.

THE BLOOD: SPINAL FLUID STREPTOMYCIN RATIO AND ITS VALUE IN THE PROGNOSIS IN TUBERCULOUS MENINGITIS. M. GOMIRATO-SANDRUCCI, Minerva pediat. **5**:993 (Nov. 30) 1953.

From data from the cases of 35 patients being treated by streptomycin for tuberculous meningitis, the author has observed that the streptomycin percentage in the blood is several times higher than the streptomycin content of the spinal fluid of patients showing clinical improvement; on the other hand, when the blood percentage is not more than two times that of the spinal fluid, the patients are doing poorly and usually die. The streptomycin blood: spinal fluid ratio should offer a useful prognostic criterion as to the course of the disease.

HIGGINS, Boston.

THE IMPORTANCE OF RECOGNIZING THE PRODROMAL SIGNS OF TUBERCULOUS MENINGITIS. A. MALLARDI, Minerva pediat. **6**:105 (Feb. 15) 1954.

As the early treatment of patients with tuberculous meningitis permits of a better response to treatment, it is important to recognize the prodromal signs of the disease. The classic prodromal signs are loss of weight (wasting), asthenia, and a change in personality. Less frequent signs are headaches, subnormal temperature, inconstant pain, vomiting and other digestive tract upsets, sleepiness, hemiparesis, vasomotor disturbances, and sporadic vertigo.

HIGGINS, Boston.

THE HYDRAZIDE OF ISONIAZID-STREPTOMYCIN TREATMENT OF TUBERCULOUS MENINGITIS. A. GENTILI and A. PACI, Minerva pediat. **6**:145 (March 15) 1954.

The conclusion is drawn that the combination of the hydrazide of isoniazid and streptomycin is superior to streptomycin and *p*-aminosalicylic acid (PAS) in the treatment of patients with tuberculous meningitis. There was a higher percentage of cures, the cures were more rapid, the early acute period of the illness was shortened, and, therefore, there were fewer neurological and auditory complications.

HIGGINS, Boston.

THE LIMITED VALUE OF PHRENIC COLLAPSE THERAPY IN THE TREATMENT OF PULMONARY TUBERCULOSIS IN CHILDREN. F. MARGSTAHLER, Minerva pediat. **6**:160 (March 15) 1954.

Fifty children with pulmonary tuberculosis at the Vialba Sanatorium have, during the past 20 years, been given pulmonary collapse treatment either by cutting (14 cases) or by crushing (36 cases) the phrenic nerve. The therapeutic results have been good in 12, questionable in 18, and of no value in 20 cases. The author is somewhat skeptical as to the real value of this procedure. With the advent of chemotherapy and of antibiotics and of the more recent methods of collapse therapy, the use of phrenic collapse has rarely been found necessary in recent years.

HIGGINS, Boston.

A STATISTICAL AND CLINICAL STUDY OF 65 CASES OF TUBERCULOUS MENINGITIS ADMITTED TO THE CHILDREN'S HOSPITAL IN TRIESTE DURING THE FIVE-YEAR PERIOD 1947-1952. T. PERI and F. FUMI, Minerva pediat. **6**:206 (March 31) 1954.

Twenty-eight of 65 children treated for tuberculous meningitis at the Children's Hospital of Trieste over a five-year period have died. The mortality rate, however, fell from 80% in 1947 to 17% in 1952. The average duration of treatment before the spinal fluid became normal dropped from 24 months in the 1947 cases to 9½ months in the 1952 cases. Six of the surviving

children have major permanent residual defects; two are deaf, one has urinary incontinence, one mental retardation and a progressive muscular dystrophy, one hemiparesis, and one cataracts and uveitis.

The daily giving of 30 mg. of streptomycin by cisternal puncture, especially during the first three months of treatment, was found to be safe and of value, in that no block to the free flow of spinal fluid occurred.

Ultimate good clinical results largely depend on the early institution of treatment. No child in this group survived whose treatment was begun later than the 14th day after the first meningeal symptoms.

HIGGINS, Boston.

THE BLOOD PICTURE OF PATIENTS UNDERGOING TREATMENT WITH ISONIAZID FOR TUBERCULOUS MENINGITIS. G. RICCI and T. COPAITICH, Pediatria **61**:849 (Nov.) 1953.

Blood cell counts were made on 20 children with tuberculous meningitis before and during treatment with isoniazid. Seventeen of the patients were cured, and three died.

No striking or consistent changes were detected in the red blood cell counts or in the hemoglobin traceable to the drug itself. However, both the red cell count and the hemoglobin showed an increase as patients with initial low counts improved in health, and similarly decreased in those dying from the disease.

The trend with the white blood cells was toward a neutropenia and a lymphocytosis, with the total count showing little if any change. Arneth's counts tended to shift to the left.

HIGGINS, Boston.

TUBERCULOUS MENINGITIS TREATED WITH STREPTOMYCIN ASSOCIATED WITH ISONIAZID. F. RAGAZZINI, M. GIUSTI, and R. COCOHI, Riv. clin. pediat. **51**:110 (Feb.) 1953.

The authors have made a clinicostatistical comparative research on various cases of meningitis in the Childrens Hospital of Florence to indicate the differential data in the course of meningitis when treated only with streptomycin (associated with paraaminosalicylic acid [PAS] and sulfone, oral and parenteral), and with streptomycin associated with isoniazid by local and oral and parenteral administration.

From the data thus collected, the authors observed that this association seems to produce a further decided improvement in the prognosis of the disease, the course of which is also remarkably shortened.

FROM THE AUTHORS' SUMMARY.

A CASE OF CONGENITAL SYPHILIS DIAGNOSED AT AUTOPSY AND ITS MEDICOSOCIAL IMPORTANCE. JULIO ESPINOZA and ALFREDO DABANCENS, Rev. chilena pediat. **24**:17 (Jan.) 1953.

The authors report a case of a 40-day-old girl who, severely ill, was brought to the first aid station, where septicemia was diagnosed; she died 48 hours afterwards. The autopsy revealed congenital syphilis, after which the syphilis in both parents was diagnosed and treated. The authors comment on the difficulty of diagnosing syphilis only by physical examination and stress the importance of educating the patient in the importance of completing his specific treatment, thus avoiding contagion. Finally, they insist on the necessity of investigating the possibility of syphilis in the pregnant mother so as not to lose the opportunity of treating both mother and fetus.

FROM THE AUTHORS' SUMMARY.

PROGNOSTIC VALUE OF THE CEREBROSPINAL FLUID IN TUBERCULOUS MENINGITIS. JORGE E. HOWARD and MANUEL ASPILLAGA, Rev. chilena pediat. **25**:1 (Jan.) 1954.

The authors studied the evolution of the cerebrospinal fluid in 107 cases of tuberculous meningitis treated with streptomycin with an aim to determine prognosis. They analyzed statistically the different values obtained in serial examinations done twice a month for the first 3 months, and monthly thereafter until 12 months. They concluded that due to the great dispersion of values found, the cerebrospinal fluid is useless as a prognostic guide in an individual case, but that, in general, those children that will die on admission to the hospital and afterwards during the whole evolution of the disease, have fluid that is more abnormal. No single component of the cerebrospinal fluid has greater prognostic value than any other.

FROM THE AUTHORS' SUMMARY.

TUBERCULOUS MENINGITIS IN THE CHILD. ROBERTO INFANTE, MARIO GONZALEZ, and ALFONSO COSTA, Rev. chilena pediat. **25**:55 (Feb.) 1954.

Isoniazid by mouth is an efficient drug in the treatment of tuberculous meningitis in the child. In doses of 7 to 10 mg. per kilogram of body weight, it was very well tolerated and did not show any toxic action. Isoniazid given exclusively by mouth in the treatment of tuberculous meningitis proved very efficient and is to be recommended in some older children if strictly controlled.

In the child less than 3 years old, the better treatment is the association of isoniazid with general and intrathecal streptomycin. This association permits the reduction of high doses of streptomycin.

The use of isoniazid has been followed by the reduction of mortality in the Roberto del Rio Children's Hospital from 69% to 10% in tuberculous meningitis.

FROM THE AUTHORS' SUMMARY.

ISONIAZID IN PULMONARY TUBERCULOSIS. OSCAR PEREZ, SERGIO IBANEZ, and JOSÉ LEGARRETA, Rev. chilena pediat. **25**:71 (Feb.) 1954.

Isoniazid was used in the treatment of 40 cases of primary pulmonary tuberculosis without observable toxic symptoms due to the drug. In 11 mild cases good results were obtained. In 27 moderately advanced cases, the results were also satisfactory. In two advanced cases a temporary improvement was obtained, but a later recurrence of the disease made the use of streptomycin necessary.

FROM THE AUTHORS' SUMMARY.

TUBERCULOUS NODES OF THE NECK. MARIO GONZALEZ and OSVALDO MONTES, Rev. chilena pediat. **25**:76 (Feb.) 1954.

Isoniazid is an effective treatment in cervical tuberculous adenitis. The most active intramuscular dose varies between 7 and 10 mg. per kilogram of body weight, maintained for a minimum of six months, and locally by intraganglion injections or injections through the fistulous tracts, in doses of 2 to 50 mg. Pus should be aspirated before the injection.

The most intense and precocious action is observed in the fistulous tracts which heal, in 90% of the cases, before the end of 15 days of treatment. Fifty-five percent of the children healed completely, and 18% showed a great improvement. Twenty-seven percent of the patients had a relapse or the results were mediocre. Ninety per cent of the patients showed considerable improvement of their general state of health with better appetite and a gain of weight.

FROM THE AUTHORS' SUMMARY.

Diseases of Blood, Heart and Blood Vessels, and Spleen

CARDIAC DYNAMICS AND THE DIAGNOSTIC ROLE OF CARDIAC CATHETERIZATION. ROBERT A. BRUCE, Northwest Med. **53**:577 (June) 1954.

This article gives an excellent review of the present status of cardiac catheterization; it lists the objectives, the indications, and the contraindications for doing the procedure, and the hazards of the technique employed.

It is highly recommended reading for the pediatrician or practitioner who has an interest in the diagnosis of congenital heart disease. In terms familiar to these practitioners, the author corrects misunderstanding and confusion which have arisen regarding information to be obtained by cardiac catheterization. He further provides background which should be useful when deciding whether or not to refer a patient for the procedure.

DOCTER, Seattle.

PEDIGREE DEMONSTRATING A SEX-LINKED RECESSIVE CONDITION CHARACTERIZED BY DRAINING EARS, ECZEMATOID DERMATITIS, AND BLOODY DIARRHEA. ROBERT A. ALDRICH, ARTHUR G. STEINBERG, and DONALD C. CAMPBELL, Pediatrics **13**:133 (Feb.) 1954.

Genetic analysis of the family of a person suffering from what appeared to be secondary thrombocytopenic purpura indicated that (1) 16 out of 40 male infants died; (2) 10 of these, including the proband, were known to have suffered from draining ears, eczematoid dermatitis,

and bloody diarrhea, and (3) the pattern of deaths agrees with the assumption that a sex-linked recessive gene is responsible for the primary deviation which led to the death of these infants.

FROM THE AUTHORS' SUMMARY.

CASE OF GALACTOSEMIA IDENTIFIED IN A 4-DAY-OLD INFANT BY PAPER CHROMATOGRAPHIC TECHNIC. JEAN D. LOCKHART and ELIZABETH ROBOZ, Pediatrics **13**:211 (March) 1954.

Galactosemia was diagnosed in a 4-day-old girl by identification of the urinary sugar as galactose, using the paper chromatographic technique. The child was placed immediately on a lactose-free formula, after which the urine became sugar- and protein-free. Since the infant was fed only for a very short time on lactose-containing formula, the prognosis may be considered good. The family history includes one healthy child, one whose galactosemia was diagnosed at the age of 3½ months, and a paternal grandfather with a "milk intolerance."

Details of the laboratory technique used in the identification of urinary galactose are presented. It is suggested that, by examination of the amniotic fluid of women in whose offspring galactosemia may be suspected because of a positive family history, a diagnosis might be made at birth and the baby never be given a lactose formula.

FROM THE AUTHORS' SUMMARY.

TRANSIENT COMPLETE ATRIOVENTRICULAR HEART BLOCK OF UNKNOWN ETIOLOGY. ALFRED S. TRAISMAN and HOWARD S. TRAISMAN, Pediatrics **13**:326 (April) 1954.

Idiopathic acquired complete atrioventricular heart block is a most uncommon entity in pediatric practice. Such a case in a 9-year-old boy with an associated Adams-Stokes syndrome is reported. A review of the literature reveals very few of such cases of unknown etiology. This boy was treated with atropine, epinephrine, and ephedrine for the Adams-Stokes attacks. His complete atrioventricular dissociation reverted to sinus rhythm with first degree atrioventricular block and a right bundle-branch block pattern. Clinically, the boy is well and engaging in normal activity.

FROM THE AUTHORS' SUMMARY.

PATENT DUCTUS ARTERIOSUS IN INFANCY. ROBERT A. LYON and SAMUEL KAPLAN, Pediatrics **13**:357 (April) 1954.

Patent ductus arteriosus produces symptoms in infancy which vary widely in kind and severity. Five patients observed during the past five years have been described briefly to illustrate these variations. One infant died at the age of one month with an aneurysm of the ductus. Another patient, with atypical signs, had a severe degree of congestive failure until surgical treatment provided dramatic relief. In the other patients, symptoms were limited to retardation of growth, frequent respiratory infections, and atypical murmurs until the later months of infancy.

In spite of the variation of physical signs and of the radiologic and electrocardiographic findings, an accurate diagnosis can often be made early in life. When doubt exists, and especially in infants suffering from cardiac failure, cardiac catheterization, aortography, or angiocardiography must be employed to secure information necessary for immediate surgical treatment. Emphasis is placed upon the fact that patent ductus arteriosus can be the sole lesion causing congestive failure in infancy.

FROM THE AUTHORS' SUMMARY.

ATYPICAL MURMURS IN PATENT DUCTUS ARTERIOSUS. R. E. BONHAM CARTER and K. W. LOVEL, Great Ormond St. J. **6**:93 (Dec.) 1953.

Seven cases are presented to illustrate the presence of patent ductus without the typical murmur. Causation of different types of murmurs is discussed and diagnostic measures outlined.

McGUIGAN, Evanston, Ill.

RETICULOGRANULOMATOSIS IN CHILDREN. L. MARTONI and M. A. DINA, Clin. pediat. **35**:695 (Oct.) 1953.

The case of a 2-year-old boy was followed over a 10-month period, from the onset of symptoms until his death. The first symptom to appear was otitis media; then, in order, there were ulcerous lesions in the mouth and the anus, papular skin lesions, a facial paralysis, bone defects

in the skull and one rib, and, finally, generalized involvement of the lungs, the spleen, the liver, and the lymph nodes. There was a transition in the marrow, and the biopsy specimen showed pathological changes as the disease progressed. At autopsy, the various organs showed different types of pathological cells. The reticuloendotheliosis was manifested at various times and places by histoid cells, eosinophiles, lipoid-containing cells, and lymphomonocytes.

A second case of reticulogranulomatosis is reported in a girl 6 years of age. Bone lesions were the first to be recognized, appearing in a lumbar vertebra, the pelvis, and the skull, in that order. Later, the girl had the symptoms of diabetes insipidus and then skin and pulmonary lesions. The early cytology pointed to eosinophilic granuloma and later transformed to histiomonocytosis and cells containing lipoids.

The thought is expresssed that the Abt-Letterer-Siwe syndrome, the Hand-Schüller-Christian syndrome, and the eosinophilic granulomatosis are, in effect, merely different evolutionary manifestations of the same disease—reticulogranulomatosis.

HIGGINS, Boston.

Diseases of Gastrointestinal Tract, Liver, and Peritoneum

PEPTIC ULCER IN CHILDHOOD. BERTAM R. GIRDANY, Pediatrics **12**:56 (July) 1953.

Peptic ulcer in childhood has been considered a relatively rare disease, although sporadic reports of its occurrence have appeared. It has not usually been included in the differential diagnosis of conditions causing recurrent abdominal pain or vomiting in the pediatric patient. The data in this article indicate that peptic ulcer is of frequent occurrence and constitutes an important pediatric problem. Psychogenic elements probably play an important role in the disease. The possible relationship of peptic ulcer to cyclic vomiting is suggested.

FROM THE AUTHOR'S SUMMARY.

PEPTIC ULCERS IN CHILDREN. RALPH C. AYE, Radiology **61**:32 (July) 1953.

The author divides peptic ulcers into two types, acute and chronic. The latter is defined as one in which symptoms have been present for a minimum of two months, a niche has been demonstrated roentgenologically, and a trial of medical therapy has produced partial or complete amelioration of symptoms. The author's four patients were observed at the ages of 3 years, 10 years, and (two) at 4 years of age. All were boys. Three boys had duodenal ulcer, and one had gastric ulcer.

ANSPACH, Chicago.

DOUBLE CONTRAST STUDIES OF COLON: POLYPS IN CHILDREN. CHARLES W. YATES, Southern M. J. **46**:315 (April) 1953.

Multiple polyposis (heredofamilial), pseudopolyps (a complication of chronic ulcerative colitis), and multiple adenomatous polyps are found in children. The adenomatous polyps seldom become malignant and are sometimes self-amputating. The chief symptom is blood in the stool. Protrusion, diarrhea, and abdominal cramps may occur. Diagnosis is established by proctoscopic examination of the rectum and lower sigmoid and by roentgen study of the rest of the colon. The double-contrast technique following thorough cleansing of the bowel by oral administration of castor oil and the use of enemas is recommended.

LOGAN, Rochester, Minn.

CAROB FLOUR IN TREATMENT OF DIARRHOEAL CONDITIONS IN INFANTS. DE LA BROQUERIE FORTIER, GEORGES LEBEL, and ANTONIN FRECHETTE, Canad. M. A. J. **68**:557 (June) 1953.

Two hundred and seventy-one babies with infantile diarrhea were treated with carob flour (Arobon). Results were superior to those previously experienced with any pectin-base medication.

MCGUIGAN, Evanston, Ill.

SENSITIVITY TO ANTIBIOTICS OF STRAINS OF BACT. COLI ASSOCIATED WITH INFANTILE GASTRO-ENTERITIS. J. SMITH and W. H. GALLOWAY, Arch. Dis. Childhood **28**:30 (Feb.) 1953.

Tests have been carried out in vitro and in vivo on the effect of antibiotics on the specific types of Escherichia coli associated with infantile gastroenteritis.

The in vitro tests showed that low concentrations of chlortetracycline (Aureomycin), chloramphenicol, and oxytetracycline (Terramycin) had a marked inhibitory action on growth over a period of 24 hours at 37 C. and very much less marked action at 48 hours. Dihydrostreptomycin was less effective.

The bactericidal action of chlortetracycline, chloramphenicol, and oxytetracycline was relatively weak and was evident only in high concentrations of 25 and 50 γ per milliliter, and then only on the more susceptible strains. Dihydrostreptomycin, in the higher concentrations, had a bactericidal action on a greater number of strains.

The in vivo tests with the antibiotics have shown that suppression of the coliform flora of the intestine could be effectively obtained, these organisms being largely replaced by members of the Proteus group.

So long as oral administration was continued, the specific types of Esch. coli were held in check, but with the cessation of treatment they tended to reappear.

Antibiotic therapy did not appear to lessen the total period of infectivity with specific coliform types, though, presumably, while being employed it limited the possibility of cross infection simply by reducing the number of organisms excreted.

KELLY, Milwaukee.

DIARRHOEA AND VOMITING IN CHILDREN. D. G. HAMILTON, M. J. Australia **2**:450 (Sept. 19) 1953.

Detailed methods for restoring fluid and electrolyte balance and proper dietary management in acute gastroenteritis in childhood are presented.

McGUIGAN, Evanston, Ill.

STEATORRHEA FROM ANTIBIOTICS. F. DI RIAMONDO, M. LA PESA, and G. CONESE, Gior. mal. infet. e parassit. **5**:389 (Nov.-Dec.) 1953.

Seven young healthy subjects were given a constant hospital diet, their feces being analyzed for fat before and after chloramphenicol medication. A course of medication in which they received 2.5 gm. of chloramphenicol daily resulted in an increase on the sixth day of 35 to 170% over the amount of fecal fat found in the preliminary tests. Analyses also showed that these subjects were not able to utilize a single feeding of 100 gm. of olive oil so well as formerly while they were receiving the chloramphenicol. The appearance of the stools after chloramphenicol had been taken for several days suggested those of sprue, being loose and fatty. There was also a tendency for the subjects to have a hypoproteinemia and smoothness of the tongue. Vitamin B complex, including added B_{12}, relieved this condition somewhat but did not wholly eliminate the fat intolerance. It would appear that the sprue-like syndrome was the result of the action of the antibiotic on the fecal flora.

HIGGINS, Boston.

THREE CASES OF HYPERTROPHIC STENOSIS OF THE PYLORUS. P. CAGINI, Lattante **24**:464 (July) 1953.

Very satisfactory results followed the surgical treatment of three infants with pyloric stenosis. Although pyloric stenosis has been found rather commonly in Scandinavia, Germany, and England, it is relatively rare in Italy, only about 30 cases having been reported in the past 30 years. As to etiology, the possibility of hormonal reaction is raised, inasmuch as the male infant is exposed to a predominance of female hormones while being carried by his mother.

HIGGINS, Boston.

THREE CASES OF ABNORMALLY SHORT ESOPHAGUS IN CHILDREN. C. SÁINZ DE LOS TERREROS, B. TARACENA DEL PIÑAL, E. MARTÍNEZ ALONSO, and F. CURBERA, Acta pediat. españ. **11**:757 (Sept.) 1953.

The authors, two pediatricians and two thoracic surgeons, present case histories of three children, each with a very short esophagus. (They use the term brachyesophagus.) The cardiac end of the stomach projected into the thorax. Clinical history, x-rays, and details of the surgical procedure are discussed.

The authors believe this condition is much commoner than is ordinarily thought. They have seen the same number of cases of brachyesophagus as of pyloric stenosis over a period of three years.

ALLEN, Santa Fe, N. Mex.

CONGENITAL ATRESIA OF ESOPHAGUS WITH TRACHEOESOPHAGEAL FISTULA. B. TARACENA DEL PIÑAL, L. GONZÁLEZ-COVIELLA, E. MARTÍNEZ ALONSO, and F. CURBERA, Acta pediat. españ. **11**:776 (Sept.) 1953.

The authors present a case of esophageal atresia with a communication between the bronchus on the left side and the lower portion of the esophagus. The child was 3 days old at the time of operation. His condition was good, and he had a strong sucking reflex. An end-to-end anastomosis was successfully done, with the correction of the fistula.

The child survived 56 hours after the operation, and autopsy showed no evidence of failure of the procedure. The authors feel that the death was due to a transfusion into the superior longitudinal sinus, as death occurred shortly thereafter.

X-rays showing the position of a rubber sound in the esophagus, as well as other illustrations, are included.

The authors point out that any child with excessive, persistent foamy secretion in the fauces should be suspected of having this type of anomaly.

ALLEN, Santa Fe, N. Mex.

CYSTIC FIBROSIS OF PANCREAS. S. JULIO ESPINOZA and P. HUMBERTO RECCHIONE, Rev. chilena pediat. **24**:10 (Jan.) 1953.

The authors studied 15 cases (10 in boys and 5 in girls) of fibrocystic disease of the pancreas in children between the ages of 2 weeks and 5 years. In all these cases no clinical signs that might reveal the diagnosis were present, their study consisting of autopsy findings. A serial investigation in 86 autopsies revealed fibrocystic disease of the pancreas in 4.65%. After studying the etiology, pathogenesis, and frequency of this disease in other countries, the authors agree with the opinion that fibrocystic disease of the pancreas is a pathological diagnosis, that its clinical manifestations do not correspond to a specific nosological picture, and that the digestive or pulmonary symptoms depend on the time factor in a process that pathogenically lies in a predisposed organism.

FROM THE AUTHORS' SUMMARY.

DIAGNOSTIC VALUE OF PNEUMOPERITONEUM IN THE CHILD. ROBERTO INFANTE YAVAR and MANUEL NEIRA SALGADO, Rev. chilena pediat. **24**:67 (March) 1953.

Artificial pneumoperitoneum is a useful method for the topographic diagnosis of supradiaphragmatic and infradiaphragmatic processes. In children the technique is simple, and our experience shows that the method is innocuous, without subjective disturbances for the patient and without accidents.

The spontaneous pneumoperitoneum is a pathognomonic sign of perforation of the digestive tube. Its appearance is early and precedes peritoneal signs. Its recognition and clinical interpretation should be known by physicians working in first-aid stations.

FROM THE AUTHORS' SUMMARY.

GENESIS OF RECURRENT COLIC-LIKE ABDOMINAL PAIN IN CHILDREN (*Nabelkoliken*). K. H. SCHÄFER and M. A. LASSRICH, Deutsche med. Wchnschr. **78**:421 (March 27) 1953.

Children with, in most instances, a typical clinical picture of *Nabelkolik* were examined roentgenologically. The number examined was 260. One of the authors (M. A. L.) conducted these examinations and will report the results in detail. The most frequent finding was an inflammatory change in the region of the last loop of the ileum and of the ileocecal valve. The presence of such an inflammation is regarded as proved when the final loop of the ileum or the ileocecal valve is infiltrated and painful on pressure. Much less frequently found were ascaridiasis, abdominal lymph node enlargement, inflammation and ulcers of the mucosa of stomach and duodenum, appendical involvement, and incomplete intussusception and certain changes of the large intestine. To what extent purely functional abdominal pain, that is, *Nabelkolik* in its narrower sense, exists the future will show.

AMBERG, Rochester, Minn.

CHOLECYSTITIS AND CHOLELITHIASIS IN CHILDHOOD. R. KÜHLMAYER, Wien. klin. Wchnschr. **65**:553 (July 3) 1953.

The author reports a boy 10 years of age with cholecystitis and cholelithiasis found at operation for supposed appendicitis.

AMBERG, Rochester, Minn.

CASE OF CONGENITAL MEGACOLON TREATED BY RECTOSIGMOIDECTOMY. Y. M. FEUILLEN, Acta pædiat. belg. **7**:179, 1953.

A 7½-year-old girl, whose first symptoms of abdominal distention appeared at 5 days of age, showed considerable distention of the large bowel above the sigmoid and rectum. Sigmoid and rectum were narrow and could not be dilated by barium enema. After removal of the narrowed portion of the bowel, the distended part returned to normal, and the child was in good health six months after the intervention.

GLASER, Jerusalem.

PATHOGENESIS OF CONGENITAL MEGACOLON. A. L. M. MUSKENS, Nederl. tijdschr. geneesk. **97**:70 (Jan. 10) 1953.

On the basis of prenatal findings and observations after birth, congenital megacolon is considered as probably the result of persistence of a prenatal condition. Intestinal contents accumulate in the most distal loop of the sigmoid, which becomes heavy. The loop is suspended from a short mesenterium, and local kinking results. Treatment should be medical.

VAN CREVELD, Amsterdam.

ATRESIA OF THE SMALL INTESTINE. B. DIJKSTRA and A. H. SMOOK, Nederl. tijdschr. geneesk. **97**:1198 (May 9) 1953.

The case of a 4-day-old baby boy is described who did not pass meconium after the first day of life. On the second day vomiting started; the vomitus became bile-tinged and malodorous. On entering the hospital the baby was in poor condition, being dehydrated and with abdominal distention. Roentgenological examination revealed distended intestinal loops in the epigastrium. The patient's condition improved after parenteral administration of fluids and antibiotics and the insertion of a gastric tube to deflate the stomach. On the fifth day of life operation was performed, with the use of local and open ether anesthesia. The intestinal loop was distended to 40 cm. beyond the duodenal-jejunal flexure, where it ended as a blind sac. There was a defect of the mesentery. A side-to-side anastomosis of the two blind ends was established and a catheter inserted in the distal loop. A mobile cecum with a long appendix was found. The postoperative treatment consisted of administration of corticotropin in a dose of 3 mg. every six hours, parenteral administration of fluids and casein hydrolysates, and blood transfusions. Feeding was started two days after the operation. On the third day bowel movements appeared. Fifteen days after the operation the infant was able to leave the hospital, completely recovered.

VAN CREVELD, Amsterdam.

VOMITING IN INFANTS. T. HALBERTSMA, Nederl. tijdschr. geneesk. **97**:1395 (May 30) 1953.

The author reviews the most important causes of vomiting in infants and its treatment. In congenital pyloric hypertrophy surgical treatment is preferred in the Netherlands over time-consuming medical treatment.

VAN CREVELD, Amsterdam.

Nervous Diseases

CONGENITAL ABSENCE OF THE ODONTOID PROCESS. N. RASKIN, Dis. Nerv. System **14**:236-238 (Aug.) 1953.

The author reports the sixth recorded case of congenital absence of the odontoid process. The patient was a 28-year-old white woman who had suffered from generalized grand mal seizures since the age of 22. During 20 months of hospitalization her attacks became increasingly frequent, and she had periods of confusion and a long series of asthmatic paroxysms. She became progressively enfeebled and died in status epilepticus. Postmortem examination disclosed a platybasic skull with a small posterior fossa and a large foramen magnum, an atlas with a thin and atrophic anterior arch, and a large axis without an odontoid process. The occipital lobes were both undersized, the left being smaller than the right. In explanation of the association of malformation, it is pointed out that the embryonic order of development of the parts involved indicates that the platybasia preceded and caused the cerebral distortion. The anterior arch of the atlas in this case was firmly attached to the unusually massive axis at the point where the odontoid process should have arisen. Hence the patient could not show the usual clinical

symptoms which lead to roentgenological diagnosis of the syndrome: pain in the neck, limitation of cervical rotation, or abnormal position of the head following an accident or exercise, due to dislocation of the atlas on the axis.

BEATON, Tucson, Ariz. [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

INFANTILE CORTICAL HYPEROSTOSIS. H. G. RISCHBIETH, Clin. Rep. Adelaide Child. Hosp. **2**:130 (May-Nov.) 1953.

A case of infantile cortical hyperostosis is described and the differential diagnosis outlined. A review of the literature is included.

McGUIGAN, Evanston, Ill.

CASE OF OTITIC HYDROCEPHALUS OF INTRACRANIAL HYPERTENSION. R. H. VON DER BORCH, Clin. Rep. Adelaide Children's Hosp. **2**:156 (May-Nov.) 1953.

Severe intracranial hypertension occurred in a child who had had an earache three weeks previously that had apparently responded well to sulfonamide. The eardrum appeared normal. Because of severe symptoms and probable venous sinus thrombosis, mastoidectomy was done, revealing extensive perisinuous clotting and apparent mural thrombosis inside the sinus. Recovery was slow but after four months was almost complete.

McGUIGAN, Evanston, Ill.

RELATION OF Q-T TIME TO SERUM CALCIUM IN THE SYNDROMES OF TETANY. R. RIZZI and C. ARENA, Lattante **24**:433 (July) 1953.

Electrocardiographic and blood calcium studies were made in eight cases of rachitic tetany, in two cases of parathyroid tetany, and in one case of essential tetany. The Q-T time, i. e., the duration of the electrical reaction of systolic contraction, was found to be markedly prolonged in the two patients with parathyroid tetany, somewhat prolonged in the patient with essential tetany, slightly increased in two of the patients with rachitic tetany, and unchanged in the other six patients. When calcium chloride had been given intravenously, the Q-T time was shortened, but this change was not in proportion to changes in the biologically active calcium content of the blood serum.

HIGGINS, Boston.

ACUTE TRANSITORY ATAXIA IN YOUNG CHILDREN. P. DENYS and L. GERIN, Acta pædiat. belg. **7**:99, 1953.

The authors describe the little-known syndrome of disturbances of the equilibrium in small children occurring in conjunction with or after acute viral diseases, together with seemingly banal upper respiratory infections or without any accompanying symptoms, as a primary disease, possibly caused by a poliomyelitis virus. The symptoms occur suddenly, last from a few days to two weeks, and disappear without sequelae. The few existing literature references are discussed and 14 cases presented.

The symptoms in the reported cases were either purely cerebellar or cerebellovestibular. In five cases they occurred in the course of upper respiratory infections; in one, after chicken pox, and in one, after probable viral pneumonia. The last five cases occurred in short succession during an epidemic of poliomyelitis.

GLASER, Jerusalem.

CLINICAL STUDY OF CEREBRAL ABSCESS IN INFANTS. C. BEECKMANS, Acta pædiat. belg. **7**:263, 1953.

The case of a 9-month-old infant who was admitted because of physical and psychomotor retardation is reported. The head appeared large and asymmetrical; the fontanel was tense, and there was intermittent strabismus. The electroencephalogram showed focal signs in the left temporo-occipital region. A pneumoencephalogram showed signs of a space-occupying lesion on the left side. A staphylococcic abscess in the left temporal region was opened, drained, and treated by daily injections of penicillin and streptomycin through the drain. The child recovered, and electroencephalographic findings returned to normal.

The child's past history revealed a maternal breast infection when the infant was one month of age and pyelitis 14 days before admission.

The author considers local trauma, direct propagation of infection, and metastatic infection as etiologic agents. The presenting symptoms were intracranial hypertension, signs of infection, and neurological signs permitting localization with the aid of electroencephalogram, pneumoencephalogram, lumbar puncture, and, finally puncture of the abscess.

GLASER, Jerusalem.

Psychology and Psychiatry

TEMPER TANTRUMS IN CHILDEN. J. C. MARFATIA, Indian J. Child Health **2**:570-574 (Dec.) 1953.

A brief well-written discussion of some of the causes of temper tantrums in children is presented.

McGUIGAN, Evanston, Ill.

MENTAL HEALTH IN THE MOTHER-CHILD RELATIONSHIP. JUAN BASCH-MARIN, Acta pediat. españ. **11**:967 (Nov.) 1953.

The author, in an inaugural address to the pediatricians of Spain, discusses the findings with reference to mother-child relationships and the dangers in a poor relationship and early separation. He mentions the work of Anna Freud, Reneé Spitz, and others.

Some of his conclusions are as follows:

1. Doctors, nurses, social workers, teachers, and dieticians should at least understand the practical applications of mental health, especially in the mother-child relationship.

2. Psychiatric consultation should be made available to the staffs of health departments, clinics, and hospitals.

3. Adequate instruction should be given to medical students and to students in other disciplines who deal with people on the normal development of personality, the dynamic interaction of human behavior, group dynamics, etc.

4. The medical student should have practical work and experience in community work, family problems, the care of chronic illness in the family, etc.

5. There should be an understanding of marriage problems and some experience in premarital counciling.

6. The family physician should be the coordinator of all resources, etc., referring patients to the proper agency, etc.

7. There should be some unity of services within a community in order to avoid duplication of effort and confusion.

8. The person should develop responsibility for his own health and that of his family and community.

The author outlines the emotional and physical needs of the child in its early years and ends with an interesting story of Father Juan Gilabert Jofre and his famous sermon on the treatment of psychotic patients and the superstitious and nonsocial practices of his day. It was written that he was so dramatic that in Valencia, at least, many reforms were started. This was in 1409.

These principles should not be forgotten. Children should not only be allowed to grow up to be healthy adults but also happy ones.

ALLEN, Santa Fe, N. Mex.

SOME PROBLEMS IN THE PSYCHOTHERAPY OF CHILDREN. E. VLCEK, Pediat. listy **8**:18, 1953.

The situational aspect of the origin, cause, and psychotherapy of psychological disturbances in children is discussed from the viewpoint of the Pavlovian principle of the unity of the organism and its environment, in this case, the unity of the child and the family.

FROM THE AUTHOR'S SUMMARY.

OUR UP-TO-DATE EXPERIENCES WITH CONTINUOUS NARCOSIS. J. SVATY, Pediat. listy **8**:31, 1953.

The experiences are described in using sleep therapy in 95 children aged 5½ to 16 years, suffering from chorea minor and stammering. These have been carried out at the Second Children's Clinic since 1950.

FROM THE AUTHOR'S SUMMARY.

Diseases of Genitourinary Tract

TREATMENT OF NEUROGENIC URINARY AND FECAL INCONTINENCE IN CHILDREN. ROBERT E. GROSS, GEORGE W. HOLCOMB JR., and HENRY SWAN, A. M. A. Arch. Surg. **66**:143 (Feb.) 1953.

Experiences of several years with different approaches and technique are summarized. Before radical measures are used, a permanent and incurable incontinence not responding to neurosurgical or medical measures must be demonstrated. Anal incontinence almost always responds to a constipating diet and to a daily enema. Mild bladder laxity occasionally responds to plication of the sphincters. Suprapubic cystotomy with closure of the bladder outlet is recommended for more severe incontinence. Other treatment methods of both fecal and urinary incontinence are discussed.

YANKAUER, Albany, N. Y.

INCREASED HAEMATURIA IN NEPHRITIS DURING CORTISONE AND A. C. T. H. ADMINISTRATION. L. MICHAELS and G. WALTERS, Arch. Dis. Childhood **28**:213 (June) 1953.

Three courses of corticotropin or cortisone were given in two cases of hemorrhagic nephritis, and one course of corticotropin was given in a case of doubtful diagnosis thought to be hemorrhagic nephritis.

In each patient there was a marked increase of hematuria, as measured by the urinary red cell count, every time the hormone was given. The increase took place from one to eight days after treatment was begun.

KELLY, Milwaukee.

A. C. T. H. IN NEPHROSIS. GAVIN C. ARNEIL and H. ELLIS C. WILSON, Arch. Dis. Childhood **28**:372 (Oct.) 1953.

The value of corticotropin and cortisone in provoking diuresis in nephrotic patients has been confirmed. The effect of these hormones on various clinical and biochemical findings was investigated. The diuresis is usually evanescent, and the mechanism involved is discussed.

These results suggest that hormone therapy is at least as good as any other form of treatment at present available for nephrosis. Cortisone treatment gave better results than corticotropin; it caused diuresis more frequently, had fewer toxic effects, and was effective when given orally.

KELLY, Milwaukee.

CLASSIFICATION OF ENURESIS. E. M. POULTON and E. HINDEN, Arch. Dis. Childhood **28**:392 (Oct.) 1953.

An analysis of 200 consecutive cases of functional enuresis is presented, and a classification based on this analysis is suggested.

In over three-quarters of the children an unusually large quantity of urine was found to be excreted at night, far more, in fact, than the child ever passed in one act. Evidence is produced to show that this peculiarity either may be inherited as a physiological variant of the normal or may be acquired as the result of certain diseases or injuries.

Some account is given of enuresis resulting from abnormalities of bladder function and also of psychogenic enuresis.

Case histories are given to illustrate the various forms of functional enuresis.

KELLY, Milwaukee.

CLINICAL AND EXPERIMENTAL STUDIES AS TO THE PATHOGENESIS AND TREATMENT OF THE NEPHROTIC SYNDROME. P. DURAND and E. DE TONI JR., Minerva pediat. **5**:717 (Sept. 15) 1953.

In a long essay on nephrosis which includes a bibliography of about 375 articles, the authors include some original observations on 24 patients, most of them children, with nephrosis.

The hyperproteinuria with its high percentage of albumin is counterbalanced by a hypoproteinemia with a low percentage of albumin. The hyperproteinuria is accompanied by a hyperaminoaciduria; this indicates (1) that the tubules fail to reabsorb amino acids which have been filtered by the glomeruli, and (2) that there has been a failure of the body to build enough protein to compensate for the increased excretion. The so-called "nephrosis-peptide" is not

characteristic of nephrosis alone, but is found in cases of nephritis, especially when there is much proteinuria.

The plasma cells in the bone marrow were found to increase with the increased hypoproteinemia and hyperlipemia, but are not accompanied by a production of sufficient gamma globulin to combat intercurrent infections well.

The opinion is expressed that renal changes are the causes of the proteinuria and also indirectly play a major part in the causing of edema.

HIGGINS, Boston.

ARTERIAL HYPERTENSION DUE TO UNILATERAL ATROPHIC INTERSTITIAL NEPHRITIS AND RELIEVED BY REMOVAL OF A CONGENITALLY HYPOPLASTIC KIDNEY. P. DURAND and R. BRUNI, Minerva pediat. **5**:1073 (Dec. 15) 1953.

A 14-year-old girl for four years had had intermittent periods of headache, vertigo, vomiting, and fever; the clinical findings were arterial hypertension (190/145), angiospastic retinitis, intermittent pyelonephritis, and a slight hyperazotemia. The condition was due to a congenital abnormally small left kidney; after nephrectomy, there was a marked fall in the blood pressure and relief of the symptoms.

HIGGINS, Boston.

HYDRONEPHROSIS DUE TO MALFORMATION OF THE URETHRA. L. GUBERN SALSACHS, Acta pediat. españ. **11**:601 (June) 1953.

This is a case of an obstruction at the neck of the bladder in the posterior portion of the urethra in a boy of 19 months.

The bladder was not markedly distended, but on both sides the calyx and pelvis of the kidney were very much enlarged, as were the ureters and the urachus, the latter of which was patent for a small distance.

The details of the treatment, surgical and medical, are described. As in most cases, the combination of infection and obstruction had caused the illness. At the time of report, a year since treatment, the child was in good health.

Although the bladder was not distended, the areas around the ureters and urethra were very much engorged, and this condition added to the trouble. Pyelograms showing distended ureters and urachus are included, as well as a figure showing the distended urachus found at the time of surgical intervention.

ALLEN, Santa Fe, N. Mex.

WILMS'S TUMOR. F. H. VAN GULIK, Nederl. tijdschr. geneesk. **97**:1763 (July 4) 1953.

The author reports a case of Wilms's tumor, of which the striking features were the age of the patient (a man of 51) and the weight of the growth (10 kg.). Surgical treatment was successful.

CREVELD, Amsterdam.

Diseases of Ductless Glands; Endocrinology

EFFECTS OF GROWTH HORMONE IN DOGS. JAMES CAMPBELL, H. R. HAUSLER, J. S. MUNROE, and I. W. F. DAVIDSON, Endocrinology **53**:134 (Aug.) 1953.

In dogs given growth hormone, the quantity of protein in the blood plasma is increased and the plasma protein pattern is altered. The amount of fibrinogen is increased, as a result of increased concentration and increase in plasma volume. The amount of other globulins is increased, because of some increase in concentration and the increase in plasma volume. The quantity of red cells in the blood is not decreased, but the volume per cent of packed cells is decreased, owing to dilution by the increased volume of plasma. The activity of the clotting system is increased, since the clotting time of the blood and the prothrombin time of the plasma are decreased. A leucocytosis occurred, due to increases in neutrophile granulocytes and in stab cells.

JACOBSEN, Buffalo.

STUDY OF INTERRELATIONSHIPS OF MATERNAL AND FETAL ADRENAL GLANDS OF RATS. JACQUELINE M. JONES, C. W. LLOYD, and T. C. WYATT, Endocrinology **53**:182 (Aug.) 1953.

During pregnancy in the human or in experimental animals, secretory activity of the adrenal cortex of the mother appears to be high. Little is known about the activity of the fetal adrenal cortex, and the interrelationships of maternal and fetal adrenals have not been widely studied.

The data collected in these experiments suggest that corticotropin does not cross the placental barrier of the rat under conditions of moderate stress or moderate dosage of corticotropin but that steroids of maternal adrenocortical origin do pass the placenta and inhibit the fetal adrenal. It appears that high levels of corticotropin in the maternal rat can enter the fetus and produce a stimulation of the fetal adrenal.

JACOBSEN, Buffalo.

MODIFICATION OF "STEROID DIABETES" BY POTASSIUM. L. W. KINSELL, H. E. BALCH, and G. D. MICHAELS, Metabolism **2**:421 (Sept.) 1953.

Evidence is presented that the refractoriness to insulin which occurred in a diabetic patient who received cortisone was considerably diminished by the administration of large quantities of potassium.

KAPLAN, New York.

RADIOIODINE UPTAKE IN STUDY OF DIFFERENT TYPES OF HYPOTHYROIDISM IN CHILDHOOD. SAMUEL H. SILVERMAN and LAWSON WILKINS, Pediatrics **12**:288 (Sept.) 1953.

A method for demonstrating small uptakes of radioiodine by the thyroid gland is described. The study suggests that in some cases of cretinism iodine-accumulating thyroid tissue may be completely absent, while in others a small amount may be present. In cretins in whom a small amount of thyroid tissue can be demonstrated, the symptoms, even though appearing during the first year of life, develop more insidiously. In cases of hypothyroidism developing later in childhood, small amounts of thyroid tissue capable of taking up iodine usually can be demonstrated.

In addition to the cases of hypothyroidism in which there is an absence of the gland or marked diminution in the amount of thyroid tissue, two patients (Cases 13 and 14) with definite hypothyroidism showed a normal uptake of I^{131}. One of these patients had a thyroid gland of normal size, the other a moderately enlarged gland. They lived in regions in which there was no likelihood of deficient iodine uptake.

One patient (Case 15) with acquired hypothyroidism took up no iodine when first studied but after the injection of thyrotropic hormone showed a normal uptake. The problem of whether acquired hypothyroidism may be due to deficiency of thyrotropic hormone is discussed.

FROM THE AUTHORS' SUMMARY.

INCREASED PLASMA 17-KETOSTEROID CONCENTRATION IN CONGENITAL ADRENAL HYPERPLASIA. L. I. GARDNER, Proc. Soc. Exper. Biol. & Med. **83**:251 (June) 1953.

Data concerning the plasma concentration and the urinary secretion of 17-ketosteroids in 4 children with untreated congenital adrenal hyperplasia have been compared with the findings for 15 normal men. The values for these children were equal to or in excess of those for the men. One patient was given 50 mg. of cortisone per day by mouth, and there occurred a fall in the concentration of both the plasma and the urinary 17-ketosteroid values.

HANSEN, Galveston, Texas.

ADRENAL INSUFFICIENCY IN NEWBORN MALE INFANTS. BARNETT P. BRIGGS, EUGENE H. CRAWLEY, and VERNON L. TOOMBS, Southern M. J. **46**:351 (April) 1953.

A report is given of four early cases of the adrenogenital syndrome associated with electrolyte imbalance. Each infant has been treated with desoxycorticosterone (Doca) acetate U. S. P. subcutaneously and by pellet implantation, and by added salt in the diet. One child is being treated with cortisone. One child died of accidental salicylate poisoning, and a brief autopsy report is included.

LOGAN, Rochester, Minn.

CASE OF CONGENITAL DIABETES. MARY E. S. WYLIE, Arch. Dis. Childhood **28**:297 (Aug.) 1953.

Seven cases of diabetes mellitus with the onset in the neonatal period are reviewed.

A patient whose diabetes was diagnosed at the age of 17 days was treated with insulin and was thriving at 8 months.

KELLY, Milwaukee.

THYMUS SYNDROMES. E. GRASSO, Minerva pediat. **5**:1057 (Dec. 15) 1953.

In a dissertation on the syndromes ascribed to the thymus gland, the following observations are made and conclusions drawn: 1. The thymus gland in many sudden deaths is of normal size; it is usually normal histologically. 2. The thymus gland varies markedly in size in persons of the same age. 3. The thymicolymphatic constitution cannot be considered a nosological entity, nor can thymic death be accepted etymologically. 4. The fact that thymectomy has proved to be of benefit indicates that the thymus gland may play a role in myasthenia gravis; that disease, however, is more probably polyglandular, and its pathogenesis is not the same in all cases. 5. Thymic lymphosarcoma and acute leukemia represent two distinct diseases, although they are structurally related; there is a possibility of an eventual change from one to the other.

HIGGINS, Boston.

Diseases of Muscles, Bones, Joints, and Lymph Nodes

STUDIES IN DISORDERS OF MUSCLES: IX. GLYCOGEN-STORAGE DISEASE PRIMARILY AFFECTING SKELETAL MUSCLE AND CLINICALLY RESEMBLING AMYOTONIA CONGENITA. W. KRIVIT, W. J. POLGLASE, F. D. GUNN, and FRANK H. TYLER, Pediatrics **12**:165 (Aug.) 1953.

Three infants (siblings) with flaccid weakness of skeletal muscle are described, with postmortem findings in two. Clinically, the disease simulated amyotonia congenita (Oppenheim's disease), but the anatomic disorder in these infants consisted of excess glycogen storage, producing its main effect in striated muscle.

Muscle biopsy is essential in the differential diagnosis of the muscular atrophies of infancy.

This disorder has many similarities to the cardiomegalic type of glycogen-storage disease and may be contrasted with the more common hepatomegalic (von Gierke's) type.

Glycogen isolated from the tissues of one of these patients showed excess branching and decreased average chain length. There was an excess of a low molecular weight component in the skeletal muscle.

FROM THE AUTHORS' SUMMARY.

MYOSITIS OSSIFICANS PROGRESSIVA. RODOLFO BURDACH, Rev. chilena pediat. **24**:387 (Oct.) 1953.

The clinical and histopathological characteristics of ossified progressive miositis are shown with a bibliography, with special reference to the pathogenic theories, showing a possible relation between this disease and diseases of collagen.

The case is reported of a 3-year-old girl whose illness started in the first months of life, involving the hip muscles and then following the classic course, characterized by invasion of the cervical and dorsal muscles in an excentric and descending progress. The rapid growth is stressed, which in a two-year period led to almost complete invalidism. Associated skeletal malformation existed, such as microdactylia of the thumbs, agenesia of the first phalanxes of the first toe, and incurvation of both small fingers.

The effect of treatment with cortisone is reported which, after a favorable initial progress, was not able to stop the development of the disease.

FROM THE AUTHOR'S SUMMARY.

THREE CASES OF BONE DISEASE. L. DE LEENER and J. CONTENT, Acta pædiat. belg. **7**:76, 1953.

The case of a baby boy who died at the age of 5 weeks and suffered from "periosteal dysplasia" (Porak and Durante's disease) is described. The authors believe that this is a form of osteogenesis imperfecta, a disease which can express itself in the above-mentioned severe early form, followed by early death and not hereditary, and the osteopsathyrosis (Lobstein's disease) of late occurrence, milder course, and more favorable prognosis, transmitted on a hereditary basis.

The second case, a 6-year-old girl, presented symptoms of dysostosis craniofacialis (Crouzon's disease); with mental retardation and physical development corresponding to the age. Her mother showed signs of the disease in mild form.

The third case, a boy 4½ years of age, with Morquio's disease, was operated on for cataracts at the age of 2 years. The clinical, chemical, and roentgenological findings are presented and the differential diagnosis to Hurler's disease discussed.

GLASER, Jerusalem.

Cutaneous Diseases; Allergy

DERMATOLOGIC EMERGENCIES. PAUL A. O'LEARY, Illinois M. J. **104**:356-360 (Dec.) 1953.

Only a few dermatologic conditions are of an emergency nature. Those most frequently encountered are acute vesicular dermatitis, lupus erythematosus, urticaria and angioneurotic edema, infections, herpes zoster ophthalmicus, and stasis dermatitis with autoeczematization. Life may be threatened in lupus erythematosus and in edema of the upper part of the respiratory tract as the result of urticaria or angioneurotic edema. In the other diseases, emergency measures may prevent development of severe complications or long periods of illness, but they are not lifesaving.

FROM THE AUTHOR'S SUMMARY.

ALLERGY IN IDENTICAL TWINS. RALPH BOWEN, J. Allergy **24**:236 (May) 1953.

The author observed 59 pairs of monozygotic twins over a period of 15 years. An allergic condition was present in one or both twins. A true bilateral allergy of similar pattern occurred in only seven instances. The allergic condition existed in only one twin in 52 instances. This is in contradistinction to the concept that allergic manifestations are the result of placental transference.

The author states, "The survey of twins challenges the concept that by making maternal dietary restrictions during pregnancy, allergy is less likely to appear in the newborn."

Many of these children first experienced their symptoms as early as 2 months of age, which poses an interesting question for the proponents of maternal rejection. It was also shown that brain-wave studies in twins tend to be similar irrespective of the presence or absence of an allergic problem.

The author suggests that monozygotic twins be allowed to grow up independently and be treated as distinct persons. Encourage them to dress independently and differently, instead of having them dress identically—same color of clothes, same tie and hair ribbon, etc. He states that "Being in the same grade of school and being with one another constantly provoked a certain competitiveness, hostility, and, oddly, a certain dependence. Usually one of the twins was more dominant and the leader."

HOYER, Cincinnati.

TUBEROUS SCLEROSIS. NATAVIDAD M. ALIMURUNG, CARMEN G. LOPEZ, and ELIAS PANTANGCO, Philippine J. Pediat. **2**:10 (Oct.-Dec.) 1953.

The first case of tuberous sclerosis in the Philippines is herein reported. This case was diagnosed ante mortem and unequivocally confirmed at autopsy.

The patient was a boy 5 years of age who showed the characteristic cutaneous lesions, adenoma sebaceum, retinal tumor, cerebral manifestations, epileptic seizures, and renal tumor.

FROM THE AUTHORS' SUMMARY.

News and Comment

GENERAL NEWS

Specialists Certified by The American Board of Pediatrics, Inc.—The following physicians were certified as specialists by The American Board of Pediatrics, Inc., in New Haven, Conn., on Dec. 3, 4, and 5, 1954.

Anhalt, Herbert S., Roslyn Heights, L. I., N. Y.
Coddington, Arthur Michael, Johnson City, N. Y.
Cornfeld, David, Philadelphia.
Crawford, Mary Newell, Devon, Pa.
Dodd, Samuel Garland, Newington, Conn.
Donovan, Ruth Ursula, Brooklyn.
Doyle, Eugenie Fleri, New York.
Ellis, Rose Raquel, Bronx, N. Y.
Fantazier, Elmer F., Wilmington, Del.
Fischer, Doane, Philadelphia.
Gall, Henry, Brooklyn.
Garner, Robert Carlyle, New York.
Garver, Kenneth Leon, Verona, Pa.
Gitlin, David, Boston.
Gleason, Charles Sherman, Jr., Wareham, Mass.
Goldstein, David, Staten Island, N. Y.
Goldstein, Paul S., New Haven, Conn.
Gordon, Melvin J., Newton Centre, Mass.
Gorten, Martin K., Baltimore.
Gow, Robert Campbell, Butler, Pa.
Granger, Richard Horace, North Haven, Conn.
Green, Sidney, Waban, Mass.
Harris, George S., Jr., New Rochelle, N. Y.
Harwood, Clarence Wayne, Middleton, Conn.
Heldrich, Frederick Joseph, Jr., Baltimore.
Hindman, Sarah Mathiott, Bethlehem, Pa.
Katcher, Avrum Labe, Germantown, Pa.
Koehler, Paul Burrell, Greenfield, Mass.
Lending, Miriam, Forest Hills, L. I., N. Y.
Lum, Natalie Inge, Petersburg, Va.
Manning, John Edward, Fall River, Mass.
Mellin, Gilbert Wylie, Bethesda, Md.
Mintzer, Joseph Herman, Saratoga Springs, N. Y.
Morris, John Preston, Boston.
Neal, Robert Ford, Bainbridge, Md.
Parrott, Robert H., Silver Springs, Md.
Perlman, Anthony, Baltimore.
Powell, Albert M., Jr., Frederick, Md.
Propst, Charles Robert, Sumter, S. C.
Reckhow, Alan H., Kenmore, N. Y.
Rogers, Henry Moore, Norfolk, Va.
Romano, Henry J., Auburn, N. Y.
Schwartz, Robert, Waban, Mass.
Silverman, Benjamin K., Princeton, N. J.
Sobel, Edna H., Boston.
Spino, Pascal Daniel, Greensburg, Pa.
Stern, Gertrude S., New York.
Svigals, Morton Paul, Mount Vernon, N. Y.
Taft, George Henry, Cranston, R. I.
Venin, Bernard, Jersey City, N. J.
Wein, Geraldine Becher, Oceanside, N. Y.
Weiss, Albert Joseph, Baltimore.
Winter, William David, Jr., Westwood, Mass.
Zaontz, John Bernard, Woodmere, L. I., N. Y.

ANNOUNCEMENTS

Coming Examinations to Be Held by The American Board of Pediatrics, Inc.—The American Board of Pediatrics, Inc., will hold oral examinations in New Orleans on March 4, 5, and 6, 1955, and in Detroit on April 1, 2, and 3, 1955.

Public Health Scholarship.—The Harvard University School of Public Health has announced its Public Health Scholarship. Applications should be sent to the Secretary, Harvard School of Public Health, 55 Shattuck St., Boston 15. Several categories of workers are being subsidized.

New Officers of Western Society for Pediatric Research.—At the meeting on Nov. 20, 1954, the following officers for the Western Society for Pediatric Research were elected: president, Robert Aldrich; vice-president, James Bosma; secretary-treasurer, Robert Alway.

Benjamin Knox Rachford Lectures.—The Benjamin Knox Rachford Lectures will be delivered in Cincinnati by Dr. Wolf W. Zuelzer, of Detroit, on March 10 and 11, 1955, at 8:30 p. m., in the Auditorium of the Children's Hospital Research Foundation.

PERSONAL

Appointment for Dr. Harry Medovy.—Dr. Harry Medovy, formerly Associate Professor of Pediatrics, has been appointed Professor and Chairman of the Department of Pediatrics in The University of Manitoba, effective Sept. 1, 1954. Dr. Medovy succeeds Dr. Bruce Chown on his retirement.

Appointment for Dr. Jerome S. Harris.—Dr. Jerome S. Harris has been named chairman of the pediatrics department at Duke University School of Medicine and Duke Hospital.

Dr. Harris, professor of pediatrics and associate professor of biochemistry, succeeds Dean W. C. Davison, who is James B. Duke professor of pediatrics and has been chairman of the department since 1927.

Books

Clinical Investigations Concerning the Problem of Birth Injury of the Central Nervous System. By Wilhelm H. Thiele. Price, not given. Pp. 52, with 9 illustrations. Ferdinand Enke, Hasenbergsteige 3, Stuttgart, W., 1954.

The author, an obstetrician, feels that all the hitherto described signs and symptoms of intracranial birth hemorrhage are of limited diagnostic value. He himself has described a syndrome that he claims to be specific for intracranial birth trauma: a cloudiness of the cornea which appears immediately after birth and may last for minutes only, but sometimes for hours or days. The cornea in these cases is not shiny and transparent but dim and milky. This phenomenon is the author's proof of intracranial trauma, and all his conclusions concerning frequency and sequelae are based on this finding, which was present in 8% of all newborns with a birth weight of more than 2,500 gm. In 1%, the birth injury was the cause of death. The surviving children, observed for two years and more, didn't show any evidence of physical and mental defects, except in some cases the presence of "neuropathic reaction." The author concludes that his observations give evidence of the high recuperating power of the juvenile nervous tissue.

As far as the prophylaxis of intracranial birth injury is concerned, all forced procedures to stimulate labor should be omitted.

Child Health and the State. By Alan Moncrieff. Price, $1.50. Pp. 48. Oxford University Press, Amen House, Warwick Sq., London E. C. 4, 1953.

In this collection of lectures bound in a 48-page book, the author briefly but adequately states the problems and the progress that has been made in Great Britain in the area of child health, stressing the position of the State in meeting these problems. "Child Health" is divided into the subjects of Infant Welfare, School Health, and the Deprived Child. It is necessary and advisable to evaluate the infant welfare program as well as the school health program from time to time to make certain that the children are getting what is really needed. World War 2 created a need for the care and welfare of children deprived of a home, parents, food, and clothing. The State partly met this need through the Children's Act of 1948. The factual information is clear and easy to read; however, the brevity detracted somewhat from the interest of reading. It is indicated that there is too great a tendency to do things for parents rather than showing them how. Health education is the best approach for the welfare of the child, thus showing parents how they can help themselves.

Newer Concepts of the Causes and Treatment of Diabetes Mellitus. Proceedings of Symposium on Diabetes Sponsored by New York Diabetes Association and held at Memorial Hospital and New York Academy of Sciences, New York City, October 8, 1953. Price, $2.50. Pp. 181. National Vitamin Foundation, 15 E. 58th St., New York 22, 1954.

This book, containing a symposium on diabetics, sponsored by the New York Diabetic Association, contains several articles of interest to the pediatrician, especially the following: "The Action of Insulin," by W. C. Stadie; "The Hyperglycemic Glycogenolytic Factor of Pancreas," by Gerald A. Wrenshall; "Indications for the Use of Various Insulins," by Franklin B. Peck; "The Management of Diabetes During Pregnancy," by David Hurwitz; "The Effects of Life Situations and Emotions Upon the Management of Diabetes," by Lawrence E. Hinkle Jr.; "The Nutritional Management of Diabetes," by Herbert Pollack. The book is printed in large type, so that it is very readable.

Eritrosedimentación: Teoriás y técnicas, su aplicación en la clínica. Edition 2. By Alberto Chattás. Price, not given. Pp. 135. Libreria y editorial "El Ateneo," Florida 340—Córdoba 2099, Buenos Aires, 1953.

The second edition of this work has been brought up to date by a thorough discussion of the various methods that have been recently developed for the estimation of the sedimentation rate and of the diseases in which some variation has been found. The history, techniques, causes for errors, and normal values are given. Diseases discussed are tuberculosis, leprosy, typhoid fever, poliomyelitis, diphtheria, respiratory disturbances, rheumatism, rheumatic fever, diseases of the

digestive apparatus, liver pathology, cardiovascular diseases, internal secretions, urinary disturbances, and surgical conditions. A final chapter is on its use in pediatrics. The author mentions several conditions, but believes the principal value is in tuberculosis, rheumatic fever, and chorea.

This is a very interesting and well-constructed book. It is written in Spanish, but it is easily read and should be of value to those interested in the subject.

Lecciones de Terapeutica Infantile. Edition 2. By Juan P. Garrahan and colleagues. Price, not given. Pp. 619, with 24 tables and 6 figures. Libreria y editorial "El Atteneo," Florida 340—Córdoba 2099, Buenos Aires, 1953.

The second edition of this very excellent textbook in Spanish, the first edition of which appeared five years ago, has been brought up to date by additions in the latest therapeutic advances and diagnostic procedures. New or more complete chapters have been added on cortisone, psychological approaches to many pediatric problems, odontology, the premature infant, and the acute abdomen.

There has been no change in the general character from the early edition, which was marked by clarity of exposition and a well-organized development of the various topics discussed. This is an excellent textbook and, although in Spanish, is easily read. It should be of great value to the pediatrician.

Mongolism. By Jacob Oster. Price, not given. Pp. 206, with 52 illustrations. Danish Science Press, Ltd., Kristianiagade 12A, Copenhagen, 1953.

This book is based on a study of over 1,000 Mongols, traced from 1925 to 1949, of which 526 were alive, from Seeland and neighboring islands in Denmark. Its object is the study of the etiology and pathogenesis of Mongolism on the basis of a large unselected series of patients and throwing light on these problems through clinical and genealogical investigations.

The author has done this in an excellent manner. All details have been included of the patients' past history and physical examinations. Many photographs have been included. Physical features are discussed with the mental. X-ray and laboratory studies have been done, and a review is given of pathoanatomical investigations reported in the literature. Oster concludes that Mongolism is not a hereditary disease, but probably has its cause in exogenous factors related to the mother's depressed reproductive faculty.

This is the largest series of cases with which the reviewer is familiar. It has been analyzed in an excellent manner and well illustrated. This is a book that should be read by all those interested in the subject.

The Child, His Parents and the Nurse. By Florence G. Blake, R.N., M.A. Price, $5. Pp. 440. J. B. Lippincott Company, 227-231 S. 6th St., Philadelphia 5, 1954.

This very understanding book is by Florence Blake, Associate Professor of Nursing Education, University of Chicago, with a foreword by Dr. Adrian H. VanderVeer, formerly Associate Professor of Pediatric Psychiatry, University of Chicago. Its nine chapters help to develop a better understanding of the parent-child relationship from the prenatal time through adolescence. By helping the nurse to this better understanding of human behavior, the book should develop in her a deeper and more sympathetic insight into the adjustment of the husband and wife to the child before and after it is born and the myriad problems which arise in dealing with the behavior of well and of sick infants and children, as well as the psychological aspects of growth, development, and maturity.

Miss Blake stresses the importance of not interrupting the child-mother (or mother-substitute) relationship, especially in illness of the child, so as not to "damage him and cripple his capacity to form and maintain relationships with others throughout his life time." Hence, the wisdom of caring for many sick children in the home if proper nursing care is available or of allowing the mother to stay with the child while he is in the hospital.

The greater part of the book is devoted to the understanding of infant and child behavior.

Inasmuch as it differs radically from the teachings in medical schools until recent years concerning infant and child behavior, this book could be read with profit by most pediatricians. It should be read by all pediatric supervising nurses and by those having responsibility for child-parent relationships.

Directory of Pediatric Societies *

INTERNATIONAL

International Union for Child Welfare

President: Mrs. Gordon Morier.
Secretary: Dr. Georges Thélin, 16 rue du Mont Blanc, Geneva, Switzerland.

IV° Congreso Pan-Americano de Pediatria

President: Dr. Leonel Gonzaga.
General Secretary: Dr. Adamastor Barbosa.
Secretariat: Rua Dias da Rocha 25, ap. 602, Copacabana, Rio de Janeiro.
Place: Rio de Janeiro. Time: 1954.

IV° Congreso Sudamericano de Pediatria, Confederacion Sudamericano de Sociedades de Pediatria

President: Prof. Carlos Prado.
Vice-President: Dr. Marcelo Garcia.
General Secretary: Dr. Alvaro Aguiar.
Place: Avenida mem de Sá 197, Rio de Janeiro. Time: 1954.

FOREIGN

Academy of Medicine, Toronto, Section of Paediatrics

Chairman: Dr. Bernard Laski, Toronto.
Secretary: Dr. Crawford Anglin, Toronto.
Place: Academy of Medicine.

Asociación de Pediatrias Españoles

President: Dr. Rafael Ramos Fernández.
Vice-Presidents: Dr. Juan Bosch Marín and Dr. Ciriaco Laguna.
Secretary-General: Dr. Luis Navas Migueloa.
Treasurer: Dr. Angel Ballabriga.

Australian Paediatric Association

President: Dr. P. A. Earnshaw, Ballow Chambers, Wickham Terrace, Brisbane. B17.
Honorary Secretary-Treasurer: Dr. D. C. Jackson, 217 Wickham Terrace, Brisbane. B17.
Place: Canberra. Time: Yearly in April.

British Paediatric Association

President: Prof. Stanley Graham.
Honorary Secretary: Dr. Reginald Lightwood, Paediatric Unit, St. Mary's Hospital Medical School, London, W. C. 2.
Honorary Secretary: Dr. Philip Evans, Hospital for Sick Children, Great Ormond St., London W. C. 1.
Place: Windermere. Time: Between Easter and Whitsunday.

Canadian Paediatric Society

President: Dr. G. R. Gayman, 710 Seymour St., Vancouver 2, B. C.
Secretary-Treasurer: Dr. J. C. Rathbun, 526 Waterloo St., London, Ont.

Danish Pediatric Society

President: Dr. A. Rothe-Meyer, Children's Hospital, Fûglebakken, Copenhagen F.
Secretary: Dr. E. Winge Flensborg, Children's Hospital, Martinsvej, Copenhagen V.
Place: Different hospitals. Time: First Wednesday of every month.

Departamento de Pediatria da Associação Paulista de Medicina

President: Dr. J. Leme da Fonseca.
Secretary: Dr. Joaquim da Costa Marques.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

DEPARTAMENTO DE PUERICULTURA E PEDIATRIA DA ASSOCIAÇÃO MEDICA DO PARANA

President: Dr. Haroldo Beltrao.
Secretary: Dr. Mucio de Ãbreu Neto.

DEUTSCHE GESELLSCHAFT FÜR KINDERHEILKUNDE

President: Prof. Dr. med. W. Keller, Freiburg/Brsg., Universitätskinderklinik.
Secretary: Prof. Dr. med. J. Jochims, Lübeck, Städtische Kinderklinik.
Place: Freiburg/Brsg. Time: September, 1955.

HELLENIC SOCIETY OF PEDIATRICS

President: Dr. Spiros Charocopos, Alexandras Ave. 1, Athens.
Secretary: Dr. Nikas Zervos.
Place: Amphitheater, Medical School of the University of Athens. Time: Once a month.

ISRAEL PEDIATRIC SOCIETY

Chairman: Prof. Sh. Rosenbaum, 26 Bialik St., Tel Aviv.
Secretary: Dr. R. Kochmann, 131 Ben Jehuda Rd., Tel Aviv.
Place: Tel Aviv. Time: April 20-22, 1955.

MELBOURNE PAEDIATRIC SOCIETY

President: Dr. H. McLorinan.
Honorary Secretary: Dr. John H. Colebatch, 12 Collins St., Melbourne.
Place: Royal Children's Hospital, Melbourne. Time: Once monthly, February to December.

NEDERLANDSE VEREENIGING VOOR KINDERGENEESKUNDE

President: Dr. G. J. Huët, 39 Brediusweg, Bussum.
Secretary: Dr. H. Reerink, % Sophia Kinderziekenhuis, 160 Gordelweg, Rotterdam.
Place: Children's Hospital or Pediatric Clinic. Time: Three times a year.

NORSK PEDIATRISK SELSKAP

President: Dr. Roald Rinvik, Children's Department, Ullevål Hospital, Oslo.
Secretary: Dr. Bjarne Andersen, Children's Department, Ullevål Hospital, Oslo.
Time: Friday, six times a year.

PEDIATRIC SOCIETY OF NEW ZEALAND

President: Dr. Edward J. Cronin, Power Board Bldg., Queen St., Auckland.
Secretary: Dr. J. D. Matthews, 104 Symonds St., Auckland.

PHILIPPINE PEDIATRIC SOCIETY

President: Dr. Renato Ma. Guerrero.
Secretary: Dr. Felix A. Estrada.
Place: Santo Tomas University Hospital and other pediatric centers. Time: Every two months.

ROYAL SOCIETY OF MEDICINE, SECTION OF PAEDIATRICS

President: G. H. Macnab, F.R.C.S.
Honorary Secretaries: Ursula James, M.R.C.P., and Leslie Scott, M.D.
Sectional Editor: D. MacCarthy, M.D.
Place: 1 Wimpole St., London W.1 (Correspondence for these honorary officers should be sent to this address). Time: Fourth Friday of each month, 4:15 p.m.

SCHWEIZERISCHE GESELLSCHAFT FÜR PÄDIATRIE

President: P. D. Dr. F. Bamatter, St.-Léger 8, Genève.
Secretary: Dr. M. Nicod, Place Bel-Air 2, Lausanne.

SOCIEDAD ARGENTINADE PEDIATRIE

President: Dr. Rudolfo Kreutzer.
Secretary: Dr. Luis M. Cucullu.
Place: Sante Fè 1178, Buenos Aires.

Sociedad Boliviano de Pediatria

President: Dr. Carlos Daza Meruvia.
Secretary: Dr. Cecilio Abela Deheza.
Place: S. C. I. S. P. Bldg. Time: Every Thursday.

Sociedade Brasiliera de Pediatria

For officers, see IV° Congreso Sudamericano de Pediatria, above.

Sociedad Chihuahuense de Pediatría

President: Dr. Alfonso Ruiz Escalona.
Secretary: Dr. Armando G. Herrera.
Place: Hospital Infantil del Estado, Chihuahua, Chih., Mexico.

Sociedad Chilena de Pediatria

President: Prof. Dr. Raúl Gantes A.
Secretary-General: Dr. Edmundo Cardemil.
Place: Medical Society, Merced 565, Santiago.

Sociedad Colombiana de Pediatria

President: Dr. Jorge Camacho Gamba.
Vice-President: Dr. Antonio Alvarez Riaño.
Secretary: Dr. Hernando Vanegas A.
Treasurer: Dr. Efrain Perry Villate.

Sociedad Cubana de Pediatria

President: Prof. Agustin Castellanos y Gonzales.
Secretary: Dr. Carlos Hernandez Miyares, Apartado 2430, Habana.
Place: Catedra de Clinica Infantil Hospital Nuestra Senora de las Mercedes and Hospital Municipal de Infancia, Habana. Time: 9 p.m., last Wednesday of every month.

Sociedad de Pediatría de Concepción (Chile)

President: Dr. Daniel Campos, Hospital Clínico Regional, Concepción.
Secretary: Dr. Obdulia Jiménez, Hospital Clínico Regional, Concepción.
Place: Hospital Clínico Regional Concepción. Time: 7:30 p.m., second Tuesday of every month, April to December, inclusive.

Sociedad de Pediatria del Litord

President: Dr. Raul Bruera.
Vice-President: Dr. Isidoro Slullitel.
Secretary-General: Dr. Elsa Galimany.

Sociedad de Pediatria y Puericultura del Atlantico

President: Dr. Atilio Marino.
Vice-President: Dr. Helena Uribe de Jimenez.
Secretary-Treasurer: Dr. Hernando Barrios-Angulo.

Sociedade de Pediatria da Bahia

President: Dr. Elysio Pereira de Atbayde.
Secretary: Dr. Eliezer Audiface.

Sociedade de Pediatria de Pernambuco

President: Dr. Antonio Aureliano.
Secretary: Dr. Claudia Figueiredo.

Sociedade de Pediatria do Rio Grande do Sul

President: Dr. Francisco Talaia de Moura.
Secretary: Dr. Enio Pilla.

Sociedade de Puericultura e Pediatria do Maranbão

President: Dr. Odorico Amaral de Mattos.
Secretary: Dr. Joao Mohana.

Sociedade Mineira de Pediatria

President: Dr. Agostinho Fernandes.
Secretary: Dr. Celio Marques Scotti.

Sociedade Portugesa de Pediatria

President: Prof. Dr. Lucio de Almeida.
Vice-President: Dr. Cordeiro Ferreira.
Secretary-General: Prof. Dr. Carlos Salazar de Sousa.
Treasurer: Dr. Mario Cordeiro.
Place: Clinica pediatrica do Hospital D. Estefania, Lisbon. Time: Monthly.

Sociedad Uruguaya de Pediatria

President: Dr. Euclides Peluffo.
Secretary: Dr. Ruben Gorlero Bacigalupi.
Place: Av. Agraciada 1464, piso 13, Montevideo.

Sociedad Venezolana de Puericultura y Pediatría

President: Dr. H. Arroyo Parejo.
Vice-President: Dr. Manuel M. Lander.
Secretary: Dr. Guillermo Tovar E., P. O. Box 3122, Caracas.
Treasurer: Dr. Carlos Lares Compos.
Place: Hospital Municipal de Niños "J. M. de los Ríos," Caracas. Time: Once a month.

Sociedad Yucateca de Pediatria

President: Dr. Humberto Perez Concha.
Secretary: Dr. Prudencio Ruiz Salazar.
Treasurer: Dr. Jorge Antiveros Andrade.

La Société de Pédiatrie de Montréal

President: Dr. Henri Charbonneau, 3095 Cherbrooke, Montréal.
Secretary: Dr. Edmond Baril, 301 Carré St-Louis, Apt. 1, Montréal.
Time: Second Thursday of each month.

Société de Pédiatrie de Paris

President: Prof. Jean Cathala, 45 rue Scheffer, Paris XVI.
Secretary: Prof. Maurice Lamy, 94 rue de Varenne, Paris VII.
Place: Hôpital des Enfants Malades, 149 rue de Sèvres, Paris XV. Time: 4 p.m., third Tuesday of each month.

Société de Pédiatrie de Québec

President: Dr. Roland Thibaudeau, 203 des Erables, Québec.
Secretary: Dr. Euclide Dechêne, 312 du Roi, Québec.
Place: Quebec. Time: May and November.

South African Paediatric Association

Chairman: Dr. Seymour Heymann, 804 Medical Centre, Jeppe St. Johannesburg.
Honorary Secretary-Treasurer: Dr. I. Kessel, Transvaal Memorial Hospital for Children, Milner Park, Johannesburg.

Swedish Association of Pediatrics

President: Prof. Sture Siwe, Lund.
Secretary: Dr. Karl-Axel Melin, Färjestadsvägen 16, Bromma.

Swedish Medical Society, Section for Pediatrics and School Hygiene

President: Docent Justus, Ström, Stockholm.
Secretary: Dr. M. d'Avignon, Pediatric Clinic, Karolinska Sjukhuset, Stockholm 60.
Place: Swedish Medical Society, Klara Östra Kyrkogata 10, Stockholm. Time: Second Friday of each month, except summer.

UNITED STATES

National

AMERICAN MEDICAL ASSOCIATION, SECTION ON PEDIATRICS

Chairman: Dr. William Weston Jr., 1428 Lady St., Columbia 1. S. C.
Vice-Chairman: Dr. Thurman B. Givan, 115 Remsen St., Brooklyn 2.
Secretary: Dr. Wyman C. C. Cole, 3001 W. Grand Blvd., Detroit 2.

AMERICAN ACADEMY OF PEDIATRICS

President: Dr. Crawford Bost, 115 Terrace Dr., San Francisco 27.
Vice-President: Dr. Harry Bakwin, 132 E. 71st St., New York 21.
Executive Secretary: Dr. E. H. Christopherson, 610 Church St., Evanston, Ill.
Treasurer: Dr. Edgar E. Martmer, 693 Washington Rd., Grosse Point 30, Mich.

SOCIETY FOR PEDIATRIC RESEARCH

President: Dr. Russell Blattner.
Vice-President: Dr. Harold E. Harrison.
Secretary-Treasurer: Dr. Sydney S. Gellis, Boston.

THE AMERICAN PEDIATRIC SOCIETY

President: Dr. Alfred H. Washburn, 2257 Ash St., Denver 7.
Vice-President: Dr. Wilburt C. Davison.
Secretary-Treasurer: Dr. Aims C. McGuinness, 1427 I St., N. W., Washington 5, D. C.
Place: Chateau Frontenac, Quebec. Time: June 15-18, 1955 (to be held in conjunction with the Society for Pediatric Research, the Canadian Paediatric Society, and the British Paediatric Association).

Sectional

INTERMOUNTAIN PEDIATRIC SOCIETY

President: Dr. John T. Mason.
Secretary: Dr. Stanley R. Child.
Place: Ambassador Athletic Club, Salt Lake City. Time: 7 p.m.

NEW ENGLAND PEDIATRIC SOCIETY

President: Dr. Reuben C. Bates, 122 Waterman St., Providence, R. I.
Vice-President (President-Elect): Dr. Louis K. Diamond, 300 Longwood Ave., Boston.
Secretary-Treasurer: Dr. Harry Shwachman, 300 Longwood Ave., Boston.
Place: Boston, except Spring meeting. Time: Wednesdays, four times a year.

NORTH PACIFIC PEDIATRIC SOCIETY

President: Dr. Carl Ashley, 2455 N. W. Marshall St., Portland, Ore.
Secretary-Treasurer: Dr. S. Gorham Babson, 1107 S. W. 13th St., Portland, Ore.

NORTHWESTERN PEDIATRIC SOCIETY

President: Dr. Stephen D. Mills, Dept. of Pediatrics, Mayo Clinic, Rochester, Minn.
Vice-President: Dr. A. J. Quellette, 1530 Larpenteur Ave., St. Paul, Minn.
Secretary-Treasurer: Dr. T. C. Papermaster, 215 Doctors' Bldg., Minneapolis.
Place: Minneapolis; St. Paul; Duluth, Minn., and Rochester, Minn. Time: January, April, July, and October.

ROCKY MOUNTAIN PEDIATRIC SOCIETY

President: Dr. Edwin Williams.
Vice-President: Dr. Oliver K. Scott.
Secretary-Treasurer: Dr. John H. Githens, Denver.

SOUTHWESTERN PEDIATRIC SOCIETY

President: Dr. Clement J. Molony, 416 N. Bedford Dr., Beverly Hills, Calif.
Secretary: Dr. Carl A. Erickson, 960 E. Green St., Pasadena, Calif.
Place: Jonathan Club of Los Angeles. Time: 6:30 p.m., second Wednesday in January, March, May, September, and November.

WESTERN SOCIETY FOR PEDIATRIC RESEARCH

President: Dr. John Anderson.
Vice-President: Dr. Robert Aldrich.
Secretary-Treasurer: Dr. Forrest Adams.

State

ALABAMA PEDIATRIC SOCIETY

President: Dr. David B. Monsky, 748 Adams St., Montgomery 5.
Secretary: Dr. Alice H. Pye, 305 Church St., Montgomery.
Place: Montgomery.

ARIZONA PEDIATRIC SOCIETY

President: Dr. Henry E. Running.
Secretary. Dr. William F. Schaffman.
Time: On Call.

CALIFORNIA MEDICAL ASSOCIATION, SECTION ON PEDIATRICS

Chairman: Dr. Joseph W. St. Geme, 511 S. Bonnie Brae, Los Angeles 5.
Secretary: Dr. Alvin H. Jacobs, 2201 Webster St., San Francisco 15.
Assistant Secretary: Dr. Clement J. Molony, 416 N. Bedford Dr., Beverly Hills.

FLORIDA STATE PEDIATRIC SOCIETY

President: Dr. Charlotte C. Maguire, 1413 Kuhl Ave., Maguire Bldg., Orlando
Secretary-Treasurer: Dr. Wesley S. Nock, 273 Alhambra Circle, Coral Gables.

GEORGIA PEDIATRIC SOCIETY

President: Dr. John A. Simpson, Athens.
Secretary: Dr. J. Harry Lange, 490 Peachtree St., Atlanta.
Place: Savannah. Time: At time of annual state meeting.

HEZEKIAH BEARDSLEY PEDIATRIC CLUB OF CONNECTICUT

President: Dr. Louis Spekter, Division of Crippled Children, Department of Health, State of Connecticut, Hartford.
Secretary: Dr. Frank E. Roth, 179 Allyn St., Hartford.
Time: With the Connecticut State Medical Society in the spring, with the Connecticut State Clinical Congress in the fall; winter meeting usually in December.

ILLINOIS STATE MEDICAL SOCIETY, SECTION ON PEDIATRICS

Chairman: Howard R. Miller, 418 Jefferson Bldg., Peoria 2.
Secretary: Harry H. Boyle, 2376 E. 71st St., Chicago 49.

INDIANA STATE PEDIATRIC SOCIETY

President: Dr. Harold D. Lynch, Evansville.
Secretary: Dr. L. T. Meiks, Indianapolis.

IOWA PEDIATRIC SOCIETY

President: Dr. P. E. Gibson, 1503 Brady St., Davenport.
Vice-President: Dr. Arlan F. Harrington, 924 M. N. B. Bldg., Cedar Rapids.
Secretary-Treasurer: Dr. Ralph E. Dyson, 3200 University Ave., Des Moines.
Place: Spring meeting in conjunction with the Iowa State Medical Society meeting. Fall meeting in Sept., at University Hospital, Pediatric Dept., Iowa City.

KENTUCKY PEDIATRIC SOCIETY

President: Dr. Margaret Limper, Louisville.
Secretary-Treasurer: Dr. Selby V. Love, Louisville.
Place: Louisville. Time: Spring.

Louisiana State Pediatric Society

President: Dr. Jack E. Strange, 3439 Prytania, New Orleans.
Vice-President: Dr. Charles E. Anderson, 1560 Line, Shreveport.
Secretary: Dr. Joseph M. Perret Jr., 2329 S. Carrollton, New Orleans.
Treasurer: Dr. William C. Rivenbark, 1448 Louisiana Ave., New Orleans.
Place: New Orleans. Time: May.

Massachusetts Hospital Association

President: Dr. Philip D. Bonnet, Administrator, Massachusetts Memorial Hospitals, Boston.
Executive Secretary: Henry G. Brickman, 14 Somerset St., Boston 8.

Michigan State Medical Society, Pediatric Section

President: Dr. Horace L. French, 301 Seymour Ave., Lansing.
Secretary: Dr. Park S. Bradshaw, 1014 Jefferson St., Muskegon.

Montana Pediatric Society

President: Dr. Orville M. Moore, 555 Fuller Ave., Helena.
Secretary: Dr. Joseph W. Brinkley, 410 Central Ave., Great Falls.
Place: Variable. Time: Spring and fall.

Nebraska Pediatric Society

President: Dr. Paul Bandroft, Lincoln.
Vice-President: Dr. Charles Tompkins.
Secretary-Treasurer: Dr. A. J. Lombardo, 401 Aquila Ct., Omaha.
Place: Children's Memorial Hospital. Time: 6 p.m., dinner, third Thursday of each month.

New Hampshire Pediatric Society

President: Dr. Mac Lean J. Gill, 1 South St., Concord.
Secretary-Treasurer: Dr. Robert F. Wilson, 113 Locust St., Dover.
Place: Alternately Concord, Hanover, Laconia, and Manchester. Time: Four times a year.

New Mexico Pediatric Society

President: Dr. M. K. Wylder, Suite 14, Medical Arts Sq., Albuquerque.
Secretary: Dr. Ly Werner, Suite 25, Medical Arts Sq., Albuquerque.
Place: Variable. Time: Every two or three months.

North Carolina Pediatric Society

President: Dr. Robert A. Moore, 1505 Elizabeth Ave., Charlotte 4.
Secretary: Dr. Fred R. Cochrane Jr., 1361 E. Morehead St., Charlotte.
Place: Pinehurst. Time: May 3, 1955.

South Carolina Pediatric Society

President: Dr. Walter Moore Hart, 117 W. Cheves St., Florence.
Secretary: Dr. John R. Paul Jr., 82 Rutledge Ave., Charleston.
Place: Columbia, S. C. Time: Second Monday and Tuesday in September.

Tennessee Pediatric Society

President: Dr. O. L. Von Canon, 846 McCallie Ave., Chattanooga.
Secretary: Dr. Oliver W. Hill, 4807 Newcom Ave., Knoxville.

Texas Pediatric Society

President: Dr. M. C. Carlisle, Waco.
President-Elect: Dr. Russell Blattner, Houston.
Secretary: Dr. James N. Walker, Fort Worth.
Treasurer: Dr. Jack M. Woodall, Big Springs.

Virginia Pediatric Society

President: Dr. T. J. Humphries, Roanoke.
Vice-President: Dr. F. R. Hopkins, Lynchburg.
Secretary-Treasurer: Dr. A. P. Booker, Charlottesville.

WISCONSIN STATE MEDICAL SOCIETY, SECTION OF PEDIATRICS

Chairman: Dr. E. H. Pawsat, Fond du Lac.
Vice-Chairman: Dr. Samuel E. Kohn, Milwaukee.
Secretary: Dr. J. R. Schroder, Janesville.
Delegate: Dr. K. J. Winters, Wauwatosa.
Alternate Delegate: Dr. F. Mellencamp, Milwaukee.

Local

ACADEMY OF MEDICINE OF CLEVELAND, PEDIATRIC SECTION

Chairman: Dr. C. T. J. Dodge, 15457 Euclid Ave., Cleveland 12.
Secretary: Dr. Beulah Wells, 10515 Carnegie Ave., Cleveland 6.
Place: Allen Memorial Medical Library. Time: 8:15 p.m., October, December, February, and March.

AKRON PEDIATRIC SOCIETY

President: Dr. Joseph M. Ulrich.
Vice-President: Dr. Lewis E. Rector.
Secretary-Treasurer: Dr. Max E. Griffin.
Time: Quarterly.

BALTIMORE CITY MEDICAL SOCIETY, PEDIATRIC SECTION

President: Dr. Wilson Grubb, 4 E. 33d St., Baltimore 18.
Secretary: Dr. Gibson J. Wells, 3101 St. Paul St., Baltimore 18.
Place: 1211 Cathedral St., Baltimore 1. Time: 8:30 p.m., second Tuesday in November, January, and March.

BRONX PEDIATRIC SOCIETY

President: Dr. M. H. Grand, 1749 Grand Concourse, New York.
Secretary: Dr. Charles T. Fried, 1220 Grand Concourse, New York.
Place: Morrisania City Hospital. Time: 8:30 p.m., second Wednesday of each month, except June, July, August, and September.

BROOKLYN ACADEMY OF PEDIATRICS

President: Dr. Stanley S. Lamm, 20 Plaza St., Brooklyn.
Secretary: Dr. Henry Cohen, 1761 E. 19th St., Brooklyn.
Place: Granada Hotel, Brooklyn. Time: 8:45 p.m., fourth Wednesday of February, March, April, October, and November.

BUFFALO PEDIATRIC SOCIETY

President: Dr. Douglas P. Arnold.
Secretary: Dr. Richard A. Downey.
Place: Children's Hospital, 219 Bryant St. Time: 8:30 p.m., first Monday of each month, September to June.

CENTRAL NEW YORK PEDIATRIC CLUB

President: Dr. Frank Williams, 58 Swan St. S., Albany.
Secretary: Dr. Fred W. Bush, 325 Park Ave., Rochester.
Place: Various cities in New York State; fall meeting jointly with Upper New York State Academy of Pediatrics. Time: Third Tuesday of April and October.

CENTRAL OHIO PEDIATRIC SOCIETY

President: Dr. O. W. Hosterman, 350 East Broad St., Columbus 15.
Secretary-Treasurer: Dr. John Riepenhoff, 2998 Sullivant Ave., Columbus.
Place: As announced. Time: Third Wednesday of September, November, January, March, and May.

Chicago Pediatric Society

President: Dr. Eugene T. McEnery.
Vice-President: Dr. Maxwell P. Borovsky.
Secretary: Dr. Ralph H. Kunstadter, 104 S. Michigan Blvd.
Treasurer: Dr. Noel G. Shaw.
Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month, October to May, inclusive.

Cincinnati Pediatric Society

President: Dr. John E. Allen, 3402 Clifton Ave.
Vice-President: Dr. Robert A. Lyon, Children's Hospital.
Secretary-Treasurer: Dr. William D. De Vaux, 7373 Brookcrest Ave.
Place: Children's Hospital, Elland Ave., Cincinnati. Time: On call.

Cleveland Academy of Medicine, Pediatric Section

Chairman: Dr. Robert Hauver, 10515 Carnegie Ave.
Secretary: Dr. William Herman, 10300 Carnegie Ave.

Corpus Christi Pediatric Society

President: Dr. C. Lewis Concklin, 2205 16th St.
Secretary-Treasurer: Dr. Sam Powell, 301 Palmero.
Place: La Louisiane Restaurant. Time: 7:30 p.m., last Wednesday of January, March, May, July, September, and November.

Dade County Pediatric Society

President: Dr. Lynn Whelchel, DuPont Bldg., Miami, Fla.
Secretary: Dr. Howard Engle, 311 Lincoln Rd., Miami Beach, Fla.

Dallas Pediatric Society

President: Dr. Floyd A. Norman, 4325 Cole Ave., Dallas.
Secretary-Treasurer: Dr. Charles L. Smith, 4207 Lemmon Ave., Dallas.
Place: Children's Hospital of Texas. Time: 6:30 p.m., first Thursday in each month.

Detroit Pediatric Society

President: Dr. Wolf Zuelzer.
Secretary: Dr. Mario S. Cioffari.
Place: Variable. Time: Five times a year.

Honolulu Pediatric Society

President: Dr. John Peyton, Medical Group, 1133 Punchbowl St.
Recording Secretary: Goonzo Yamashita, 1618 S. King St.
Corresponding Secretary-Treasurer: Dr. H. M. Sexton, Straub Clinic, 1020 Kapiolani St.
Place: At doctors' homes in rotation. Time: 6 p.m., third Thursday of each month.

Houston Pediatric Society

President: Dr. David Schrum, 5101 Fannin St., Houston.
Secretary: Dr. Juanita Thacker Hart, 306 South 7th St., Bellaire.
Place: River Oaks Country Club. Time: 6:30 p.m., fourth Tuesday of each month.

Kansas City Southwest Pediatric Society

President: Dr. Frank Houge, Plaza Medical Bldg., Kansas City, Mo.
Secretary: Dr. S. J. Kranson, Truman Rd. and Ash, Independence, Mo.
Place: University Club, Kansas City, Mo. Time: On call.

Long Beach Pediatric Society

President: Dr. Kenneth Berkaw, 211 Cherry Ave., Long Beach, Calif.
Secretary: Dr. C. W. Latshaw, 3821 Atlantic Ave., Long Beach, Calif.
Place: Victor Hugo Restaurant, 730 E. Broadway. Time: First Tuesday night of every month.

Los Angeles County Medical Association, Pediatric Section

President: Dr. Sidney Rosin, 6230 Wilshire Blvd., Los Angeles.
Secretary: Dr. Arthur J. Holmes, 65 N. Madison Ave., Pasadena 1.
Place: Los Angeles County Medical Association Bldg., 1025 Wilshire Blvd., Los Angeles. Time: Second Thursday of alternate months beginning in September.

Louisville Pediatric Society

President: Dr. Harry S. Andrews.
Vice-President: Dr. Joseph Little.
Secretary-Treasurer: Dr. Kenneth P. Crawford.
Place: Variable. Time: Five times a year, as announced.

Medical Society of the County of Kings and Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Jacob J. Lichterman, 1433 Union St., Brooklyn.
Secretary: Dr. Bernard Greenberg, 755 Ocean Ave., Brooklyn 26.
Treasurer: Dr. David Dragutsky, 1466 49th St., Brooklyn.
Place: Kings County Medical Society Auditorium, 1313 Bedford Ave., Brooklyn. Time: 8:45 p.m., fourth Monday of January, March, April, October, and November.

Medical Society of the District of Columbia, Section on Pediatrics

President: Dr. George Maksim, 1418 Good Hope Rd. S. E., Washington 20.
Secretary: Dr. John E. Cassidy, 5402 Connecticut Ave. N.W., Washington.
Place: Medical Society Bldg., 1718 M St. N. W., Washington. Time: Spring and fall.

Memphis Pediatric Society

President: Dr. Clifton W. Woolley, 2448 Poplar Ave.
Vice-President: Dr. Steve H. Turnbull, 848 Adams.
Secretary-Treasurer: Dr. Charles H. Householder, 321 S. Bellevue.
Place: Rotated among four hospitals. Time: 7:30 p.m., second Tuesday of each month.

Milwaukee Pediatric Society

President: Dr. Kenneth Winters, Wauwatosa, Wis.
Secretary: Dr. James P. Conway, Milwaukee.
Place: Milwaukee University Club. Time: Second Wednesday of February, April, June, and October.

Mobile Pediatric Society

President: Dr. Daniel Sullivan, 1420 Government St., Mobile, Ala.
Secretary: Dr. Robert O. Harris III, 1514 Government St., Mobile, Ala.
Place: Admiral Semmes Hotel. Time: 7:30 p.m., fourth Thursday of September, November, January, March, and May.

Nashville Pediatric Society

President: Dr. Thomas Weaver, Bennie Dillon Bldg., Nashville, Tenn.
Secretary: Dr. Ethel Walker, 3415 West End Ave., Nashville, Tenn.
Place: Homes of members. Time: Third Thursday of every month.

Nassau Pediatric Society

President: Dr. Mildred Wicker Jackson, 254 South St., Oyster Bay, N. Y.
Secretary-Treasurer: Dr. David J. Posner, 98 Wolcott Rd., Levittown, N. Y.
Place: Felice's, Post Ave., Westbury, N. Y. Time: 7:30 p.m., second Monday of October, December, February, April, and June.

New York Academy of Medicine, Section on Pediatrics

Chairman: Dr. Jerome L. Kohn, 50 W. 96th St., New York 25.
Secretary: Dr. Lawrence B. Slobody, 1 E. 105th St., New York 12.
Place: New York Academy of Medicine, 2 E. 103d St. Time: 8:30 p.m., second Thursday of each month, November to May, inclusive.

Northern California Pediatric Society

President: Dr. Edward Campion, 707 C St., San Rafael.
Secretary-Treasurer: Dr. Joseph H. Davis, 300 Homer Ave., Palo Alto.
Place: San Francisco. Time: Quarterly, as speakers are available.

Oklahoma City Pediatric Society

President: Dr. C. M. Bielstein, 301 N. W. 12th St., Oklahoma City.
Secretary: Dr. Louis Frank, 1200 N. Walker St., Oklahoma City.
Place: Oklahoma Club. Time: On call.

Philadelphia Pediatric Society

President: Dr. Norman Kendall, 3401 N. Broad St., Philadelphia 40.
Secretary: Dr. F. H. Harvie, 133 S. 36th St., Philadelphia 4.
Place: College of Physicians, 19 S. 22d St. Time: 8:30 p.m., second Tuesday from October to May, inclusive.

Pittsburgh Pediatric Society

President: Dr. Christian John Stoecklein, 519 Medical Arts Bldg., Pittsburgh 13.
Secretary-Treasurer: Dr. James W. Stirling, 694 Lincoln Ave., Pittsburgh 2.
Place: Western State Psychiatric Institute and Clinic. Time: Second Friday in October, December, February, and April.

Queens Pediatric Society

President: Dr. Moe Goldstein, 111-32 76th Ave., Forest Hills, N. Y.
Secretary: Dr. John Finnegan, 36-19 Bowne St., Flushing, N. Y.
Place: Seminole Club, 112th St. and 70th Rd., Forest Hills, N. Y. Time: Second Tuesday in October, December, February, and April.

Richmond Pediatric Society

President: Dr. Hobart T. Dougan, 1832 Monument Ave., Richmond 20, Va.
Secretary: Dr. Robert A. Hoffman, 1001 W. Franklin St., Richmond 20, Va.
Place: Medical College Hospital. Time: 8 p.m., third Thursday of each month, except June, July, and August.

San Antonio Pediatric Society

President: Dr. Philip Magrish, 402 Fulton Ave.
Secretary: Dr. Jack M. Wright, 403 Summit Ave.
Time: 7:30 p.m., supper, first Tuesday of each month.

St. Louis Pediatric Society

President: Dr. H. E. Wachter, 8505 Delmar.
Vice-President: Dr. Raymond J. LaDriere, 35 No. Central, Clayton 5, Mo.
Secretary-Treasurer: Dr. Leo F. Gowen, 337 W. Lockwood, Webster Groves 19, Mo.
Place: Medart's Restaurant. Time: 7 p.m., second Thursday of each month from September to May, inclusive.

Santa Barbara Pediatric Society

President: Dr. Clarence B. Hills, 22B. W. Micheltorena St., Santa Barbara, Calif.
Secretary: Dr. Louise P. Brown, 3017 Foothill Rd., Santa Barbara, Calif.
Place: Varies. Time: Two to three times yearly, on call.

Seattle Pediatric Society

President: Dr. O. William Anderson.
President-Elect: Dr. Robert Tidwell.
Secretary-Treasurer: Dr. James L. Tucker.
Place: College Club. Time: 6:30 p.m., fourth Friday of each month from September to June.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Fred J. Chapin, 101 John St., Bay City.
Secretary-Treasurer: Dr. George H. Lowrey, University Hospital, Ann Arbor.
Place: University Hospital, Ann Arbor.

Waco Pediatric Society

President: Dr. Jack Flowers.
Secretary: Dr. F. W. Hoehn.
Place: 1918 Austin Ave., Waco, Texas. Time: Third Tuesday of each month.

Westchester County Medical Society, Pediatric Section

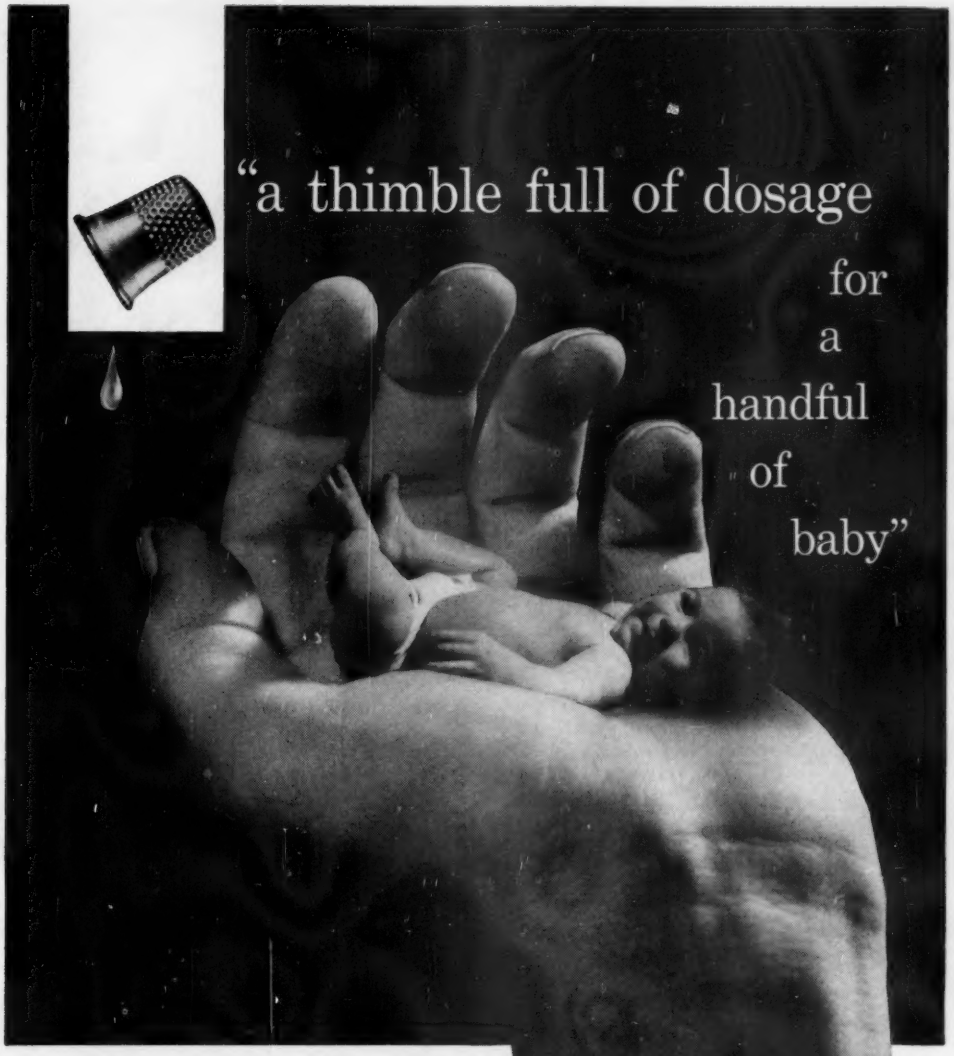
Chairman: Dr. Donald R. Reed, 14 S. Broadway, Irvington, N. Y.
Secretary: Dr. Elvira Ostlund, 64 Highland Rd., Rye, N. Y.
Place: The Roger Smith Hotel, White Plains, N. Y. Time: Four dinner meetings a year, November, February, April, and June.

Western Michigan Pediatric Society

President: Dr. A. Morgan Hill, 310 Fulton, E., Grand Rapids.
Secretary: Dr. John C. Montgomery, 1810 Wealthy St., S.E., Grand Rapids.
Place: Two meetings in Grand Rapids, two elsewhere. Time: First Wednesday of October, November, April, and May.

Wichita Pediatric Society

President: Dr. Frank L. Menehan.
Secretary: Dr. R. C. Knappenberger.
Place: Homes of members. Time: 8:30 p.m., first Thursday, October through May.



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1. Sivertsen, E.: Hvalradets Skrifter, Scientific Results of Marine Research, No. 26, Oslo, 1941.
2. Gunther, M.: Brit. J. Nutrition *6* (No. 2) :215, 1952.

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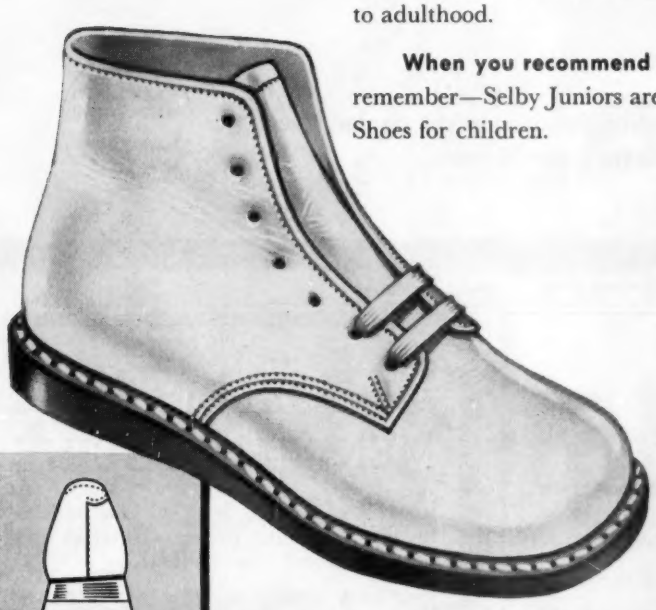

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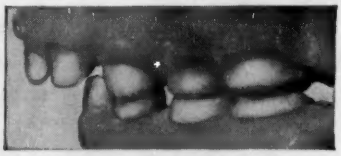

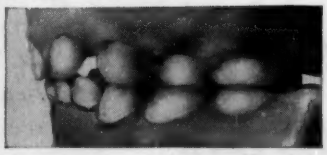

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In the organization of the low-calorie diet, adequate quantities of all the essential classes of foods—meat, eggs, milk, vegetables, fruit, enriched or whole grain bread, restored cereals—must be supplied to avoid specific nutrient deficiencies. An allowance of 400 I.U. of vitamin D should be assured.[2] By substitution of skim milk for whole milk, a 1000- to 1200-calorie diet can include an adequacy of all essentials for children 10 to 14 years of age.[1]

The protein of the diet, at least 1 gram per pound of average weight for age of the child, should come largely from animal sources.[1] The dietary fat preferably should constitute, by weight, one fourth that of the carbohydrate and not more than one half. The foods should provide as much bulk as possible.

A suggested low-calorie diet includes 2 ounces of lean meat in the noon and evening meals[1] and liver once a week. Lean meat provides large amounts of anabolically effective protein, of B vitamins, and of iron, phosphorus, and potassium. Liver, in particular, constitutes an outstanding source of vitamin A, riboflavin, niacin, vitamin B_{12}, folic acid, and iron.[3]

---

1. Nelson, W. E.: Obesity, in Nelson, W. E.: Textbook of Pediatrics, 6th ed., Philadelphia, W. B. Saunders Company, 1954, pp. 306-307.
2. A Report of the Food and Nutrition Board: Recommended Dietary Allowances, Washington, D. C., National Academy of Sciences—National Research Council, Publication 302, 1953.
3. Sherman, H. C.: Chemistry of Food and Nutrition, 8th ed., New York, The Macmillan Company, 1952.

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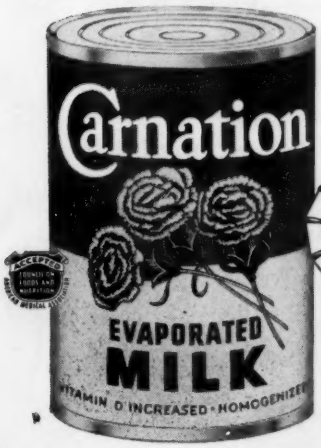

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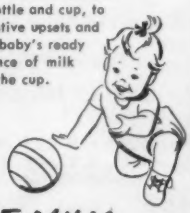

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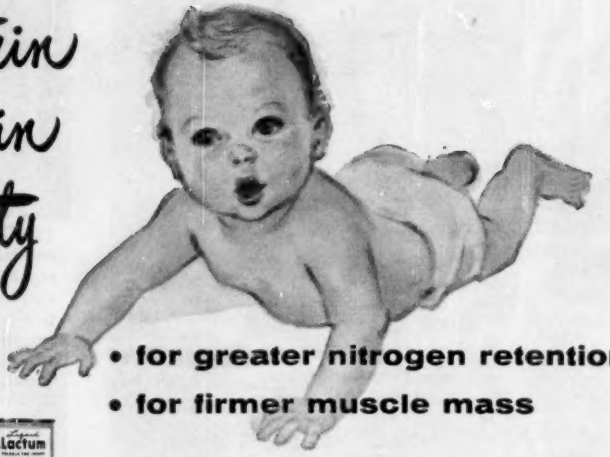

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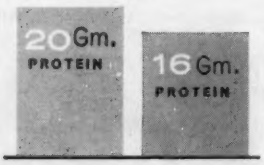

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1. Jeans, P. C., in A.M.A. Handbook of Nutrition, Philadelphia, Blakiston, 1951, pp. 275-298. 2. Stare, F. J., and Davidson, C. S., in The Proteins, American Medical Association, 1945.

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